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ICRDB CANCERGRAM

DIETARY ASPECTS OF CARCINOGENESIS

SERIES CK03 No. 82/01 JANUARY, 1982

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STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. MUTAGENIC ACTIVITY OF CAFFEINATED AND DECAFFEINATED COFFEE.

Kam JK
Univ. California, Berkeley, CA, 94720
Cancer Detect Prev; 3(2):507-511 1981

Two types of coffee (caffeinated and decaffeinated) were tested for their mutagenicity using bacterial strains using TA98 and TA100 in Ames Salmonella/Microsome test. According to the Ames criteria, both were found to be mutagenic in the ab-

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sence of S-9. The mutagens did not require liver enzymes to be activated. (Author abstract) (13 Refs)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

2. EFFECTS OF COOKING AND/OR PROCESSING UPON LEVELS OF AFLATOXINS IN MEAT FROM PIGS FED A CONTAMINATED DIET.

Furtado RM, Pearson AM, Gray JI, Hogberg MG, Miller ER
(c/o Pearson) Dept. Food Science and Human Nutrition, Michigan State Univ., East Lansing, MI, 48824
J Food Sci; 46(5):1305-1308.1319 1981

Pork loins, bellies and hams from pigs fed a diet containing 571 and 386 ug of aflatoxins B1 and B2 per kg of feed, respectively, for 42 days were analyzed for aflatoxins in the fresh, fresh-cooked, cured smoked (hams only) and cured-smoked-cooked states. Although some destruction of aflatoxins B1 and B2 occurred during cooking and/or processing, the max amount of inactivation did not exceed 41% of the total, and was generally in the range of 15-30%. Thus, results suggest that aflatoxins are quite stable during cooking and/or processing. (Author abstract) (26 Refs)

3. DETECTION OF CHLOROFORM IN THE TISSUES OF FRESHLY EVISCERATED POULTRY CARCASSES EXPOSED TO WATER CONTAINING ADDED CHLORINE OR CHLORINE DIOXIDE.

Robinson D, Mead GC, Barnes KA
Agricultural Res. Council Food Res. Inst., Colney Lane, Norwich NR4 7UA, England
Bull Environ Contam Toxicol; 27(2):145-150 1981

Possible risks related to use of superchlorinated water in food processing were investigated. Twenty-eight freshly-eviscerated broiler chickens were treated according to processes used commercially to reduce bacterial counts and prolong shelf life. Chickens were (1) sprayed with 1.5 liter (L) water/carcass containing 20mg/L free chlorine from hypochlorite; (2) immersed for 5 min in 2.5 L of water per carcass containing 5 mg/L ClO2 or 50 mg/L free chlorine at 15-16 C;(3) immersed for 20 min in 2.5 L per carcass containing either 5 mg/L ClO2 or 5-50 mg/L free chlorine at 1.5-1.5 C; or treated by all three methods, using only free chlorine, followed by cooking. Chloroform levels in skin, fat, and muscle were highest (144, 447, and 177 ug/kg, respectively) in chickens treated by treatment 2. Chilled temperatures reduced the chloroform levels when either ClO2 or free chlorine was used. ClO2 use achieves desired bacterial levels at much lower concentrations than free chlorine does. However, data suggest that the risks from chloroform in chilled-stored chickens is not significantly greater than the risk from chlorinated drinking water. (10 Refs)

4. ASBESTOS, AN ENVIRONMENTAL POLLUTANT.

Zamfir G, Diaconu R
Institutul de igiena si sanatate publica, Iasi, Romania
Igiena; 30(1):1-9 1981

Studies on the carcinogenic effect of asbestos are reviewed. One hundred and eighty-nine cases of cancer were found among 623 workers exposed to asbestos for more than 20 yr in New York and New Jersey against an expected mortality of 47.2. Lung cancer was found in 84 cases, pleural mesothelioma in 8, peritoneal mesothelioma in 24, cancer of the stomach, colon, rectum, and esophagus in 41, and cancer of other localizations in 32. Positive correlation was found between digestive tract cancer mortality and the product of exposure. There is no conclusive evidence of increased incidence of pleural and peritoneal mesothelioma due to ingestion of drinking-water containing asbestos. (39 Refs) (In RUM)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

5. NEW CONCEPTS IN NUTRITION AND CANCER: IMPLICATIONS FOR FOLIC ACID.

Butterworth CE
Dept. Nutrition Sciences, Univ. Alabama, Birmingham, AL, 35294
J Dent Child; 48(4):300-303 1981

Many of the proposed mechanisms by which diet and nutrition may be related to cancer seem to involve a genetic or chromosomal injury that may be conditioned by nutritional factors such as folic acid (FA). Hormone-induced localized nutrient deficiency has been postulated as a mechanism involved in the development of cervical dysplasia in women taking oral contraceptives (OCs). In a clinical trial, FA supplementation led to the regression of OC-induced cervical dysplasias in women who continued to take OCs. No such regression occurred in placebo-treated women, some of whom developed carcinoma in situ. The concept of nutritionally dependent fragile sites and somatic mutations on human chromosomes is not supported by clear evidence that chromosomal fragility is involved in carcinogenesis. However, a combination of chromosomal fragility and deficiencies of FA or another essential nutrient may account for specific mutations in target tissues. One published report describes 34 cases of serious hematologic disease (including 5 acute leukemias) in a family with an inherited defect of cellular FA uptake. Cervical cancer is circumstantially linked to a DNA virus (herpesvirus), and several RNA viruses are known to be oncogenic in animals. The retroviruses induce RNA-directed DNA synthesis via reverse transcriptase. There is good reason to suspect that FA-containing coenzymes play an important role in this DNA synthesis, as they do elsewhere. Folacin plays an important role in the infection of Escherichia coli by T-even bacteriophages. FA and its analogs may play a similar role in viral oncogenesis. (17 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

6. INHIBITION OF N-NITROSAMINE FORMATION BY SOYA PRODUCTS.

Kurechi T, Kikugawa K, Fukuda S, Hasunuma M
Tokyo Coll. Pharmacy, 1432-1, Horinouchi, Hachioji, Tokyo 192-03, Japan
Food Cosmet Toxicol; 19(4):425-428 1981

The soya products Tofu (soya bean curd), Miso (soya bean paste), soya milk without additives, and Bonlact (powdered soya milk with linoleic acid, vitamins, and minerals added) were tested at pH 3, 4, and 5. Known amounts of the soya products were added to reaction mixtures containing sodium

citrate buffer (pH 3 or 4) and NaNO2. All of the products tested reduced the levels of nitrite in the reaction mixtures. Reductions were faster at pH 3 than at pH4 and were dependent on the amount of soya product present. The most potent product was Miso, followed by Bonlact, Tofu, and soya milk, in order. Soya products were added to mixtures of NaNO2 and dimethylamine or NaNO2 and diethylamine. All products inhibited the formation of N-nitrosodimethylamine with greatest inhibition at pH 3. The most potent product was Bonlact, followed in order by Miso, Tofu, and soya milk. The in vitro results suggest that soya products may inhibit the formation of the potential carcinogens, the n-nitrosamines, in the stomach. The component(s) which causes the inhibition is not known. It may be either the unsaturated fatty acid component of the lipids or any of a variety of phenolic compounds. (19 Refs)

7. ENDOGENOUS CARCINOGENS.

Hill MJ
Bacterial Metabolism Res. Lab., Central Public Health Lab., London, England
Practitioner; 225(1356):803-804,807-809 1981

Estradiol is a potential endogenous carcinogen. Consuming a high fat/high energy diet, by increasing circulating estrogen levels and estrogen receptor activity, may increase the risk of carcinogenesis in estrogen-dependent tissues (breast, endometrium, possibly ovary). The role of of cholesterol in carcinogenesis is not yet clear. The author's studies showed no relationship between fecal cholesterol levels and the risk of intestinal cancer in case-control studies. Some of the numerous urinary metabolites of tryptophan seem to be related to non-industrial bladder cancer. Intestinal bacteria can derive carcinogenic substances (ethionine, phenol, p-cresol, N-nitroso compounds, bile acid metabolites, etc) from dietary components and convert exogenous chemicals (eg, polycyclic aromatic hydrocarbons) to proximate carcinogens. It is relatively easy to determine which carcinogens might be produced under ideal conditions, but there is limited information on which carcinogens are actually produced in vivo, and very little information regarding their importance. (27 Refs)

8. EPIDEMIOLOGICAL ASPECTS OF ESOPHAGEAL CANCER.

Ruggieri M, Tosato F, De Rocco K, Lepore M, Cipriano E, Paolini A
IV Cattedra di Clinica Chirurgica Generale e Terapia Chirurgica, Universita degli Studi di Roma, Rome, Italy
Recenti Prog Med; 70(5):545-556 1981

The epidemiology of esophageal cancer (EC) is reviewed and epidemiological data are presented for a series of patients subjected to surgery for EC at a clinic during the period 1971-1980. The factors probably favorable for the development of EC are ethyl alcohol, tobacco, polycyclic aromatic hydrocarbons (benzopyrene, dibenzanthracene, and 3-methylcholanthrene) present in smoked foods, nitrosamines, molybdenum, tannin, hot food, diet deficient especially in thiamine, riboflavine, and vitamin C, esophageal stenosis, peptic reflux esophagitis, brachyesophagus, achalasia of the cardia, gastric resection, and Plummer-Vinson syndrome. The epidemiological analysis of the patients treated during the 1971-1980 period showed that all patients were in the age bracket 42-75 with a mean of 59.3 yr, and that women accounted for 5.2% of the patients only. Alcohol consumption (1 liter/day) was established in 18.5% of the patients, alcohol consumption and smoking (10 or more cigarettes/day) in 52.5%, smoking only in 18.5%, and neither smoking nor alcohol consumption in 10.5%. The cervical third of the esophagus was affected in 10.5% of all cases, the thoracic third in 34%, and the abdominal third in 55.5%. (28 Refs) (In ITA)

9. THE GASTRIC CANCER IN CHIRIQUI - FREQUENCY AND SOME EPIDEMIOLOGIC DATA.

Arosema A L
Panama
Rev Med Caja Seguro Soc; 13(1):160-165 1981

Panama is situated between Colombia and Costa Rica both countries with high incidence of gastric cancer. Panama's province of Chiriqui (C) is bordering on Costa Rica. The gastric cancer rate for C was 3.49/100000, this was the av for the three yr studied (1975-1977). The reported values for Costa Rica were 34/100000 for women and 64/100000 for men, which are similar to those found in Chile and not too far behind Japan with the highest values in the world. The frequency and the epidemiologic data for C coincide with other countries except for the blood group, which was predominantly O. Matson and Swamson reported that A and B blood groups probably belonged to European descendents and the O blood was predominant in the American native population, which constitute the majority in the C province. The areas of high frequency of gastric cancer in Colombia and Costa Rica are mostly settled by European descendents. The low frequency of gastric cancer in C when compared with high values found in neighboring Costa Rica are unexpected. The environmental factors, which are suspected to influence the high rate of gastric cancer, are found in C like: high mineral content of some areas especially copper, the volcanic zone and high consumption of smoked meats. The suspected protective environmental factors are also present: low altitude, adequate consumption of milk, fresh fruit and vegetables, better conservation of food by refrigeration and the predominant O blood group of the native population. (10 Refs) (In SPA)

10. EPIDEMIOLOGY OF GASTRIC CANCER IN CHILE.

Armijo R, Detels R, Coulson AH, Medina E, Orellana M, Gonzalez A
Div. of Epidemiology School of Public Health, Univ. of California, Los Angeles, CA, 90024
Rev Med Chil; 109(6):551-556 1981

Japan has the highest gastric cancer mortality (GCM), followed closely by Chile and Costa Rica. The GCM in Chile was studied in 1957-1975 period. The extreme south and north had the lowest values for GCM were 20/100,000, which increases as approaching the center. The highest values for GCM were 50/100,000, found in 3 agricultural provinces 200 kilometers south of Santiago. The GCM decreased slightly worldwide between 1957 and 1972, this is true for Chile, except for the high risk areas. Chile has vast reserves of natural nitrates and they are used widely as fertilizers. Gastric cancer patients in controlled, matched studies were shown to have been residents longer in the areas of high nitrates utilization. Nitrates and nitrites were measured in saliva and urine of urban children, and edible vegetables of different areas, but no significant differences could be found. All factors, which could influence the results are analyzed and discussed. (10 Refs) (In SPA)

PUBLIC POLICY CONCERNING DIETARY ASPECTS OF CARCINOGENESIS

11. HORMONES AND ANTI-BIOTICS. A REVIEW OF THE USE AND EXISTING CONTROLS, DANGERS AND TOXICOLOGY OF THESE SUBSTANCES IN ANIMAL HUSBANDRY AND FOOD PRODUCTION.

Bureau Europeen des Unions de Consommateurs
29 rue Royale, 1000 Brussels, Belgium
Br Food J; 83(902):68-71,79 1981

A report on the use of hormones and antibiotics for growth promotion in animal husbandry is reviewed. Administration of the potential carcinogen diethylstilbestrol, considered the most efficient growth-promoting steroid, is controlled to varying extents and with varying success in the European Economic Community (EEC) member states. National reactions to the appearance of hormones in the food chain, methods of testing, national legislative controls, and a proposed pan-EEC regulation forbidding use of all sexual hormones and antihormones for growth promotion are detailed. (1 Ref)

12. REGULATION OF CARCINOGENS IN FOOD.

Rodricks JV
Clement Associates, Washington, DC, 20007
Ann NY Acad Sci; 363:29-35 1981

Food chemicals include naturally occurring chemicals, food additives, and food contaminants. Easiest to eliminate are additives, followed by contaminants and naturally-occurring chemicals. The public perceives the risks associated with these food chemicals in different ways: additives are seen as most threatening, contaminants next, with little thought given to naturally-occurring chemicals. These three classes of food chemicals are also treated differently by law. Food additives must acquire premarket approval from the Food and Drug Administration. Dangerous food contaminants must be shown by the government to be injurious to health before action can be taken. Present law allows the the striking of some balance between the benefits of human health protection and the costs of destroying food contaminated by a particular substance. Natural food components have been largely ignored by law. A health risk must first be demonstrated, then only in the event of a clear and obvious benefit to the public health can elimination of the food be considered. No methodology permits the absolute assurance of safety for a finite level of exposure to any substance displaying any form of toxicity. An operational definition of residue must be determined. A risk-based standard is proposed. Measurement of risks and determination of how much risk can be accepted are discussed. The question of 'acceptable' risk is addressed. (no Refs)

13. REGULATION OF CARCINOGENS IN FOOD: DISCUSSION.

Hopper PF
General Foods Corporation, White Plains, NY, 10625
Ann NY Acad Sci; 363:37-41 1981

The risk of potentially carcinogenic constituents in the food supply is dealt with by ignoring some of the risks and by performing some sort of risk-benefit assessment of the substance. The benefit of undertaking comprehensive and useful risk-benefit assessment is emphasized. The Delaney clause, which prohibits an additive if it poses a risk of cancer promotes the idea that no benefit shall be deemed to justify any demonstrable risk of cancer, no matter how small. This clause has been modified to permit the use of drugs or substances in animal feeds provided that the substance will not adversely affect the animal and no residues will be found in the edible tissues. While in the past, substances with a risk were dropped in favor of a substance that appears to be less risky, in some areas alternatives are limited. Risks and benefits must be compared more carefully. In addition, many substances once thought harmless have recently been proven to be of some risk. The statutory and administrative changes proposed by the National Academy of Sciences and the Food Safety Council are discussed. Risk/benefit comparisons will have to be restricted, not broad, leaving any decisions to the consumer. The risks inherent in the natural food supply must be studied more precisely and other risks that may reduce these or add to them only in small and tolerable ways must be accepted. (no Refs)

14. THE ROLE OF LAW IN DETERMINING ACCEPTABILITY OF RISK.

Green HP
Fried, Frank, Harris, Shriver and Kampelman, Washington, DC
Ann NY Acad Sci; 363:1-12 1981

The role of the law in determining the acceptability of risks with respect to potential or known carcinogens is addressed. The manner in which the common law handles the acceptability of risk is discussed. Legislation and regulatory agencies are reviewed. Several examples illustrating the approach taken to safety regulations involving carcinogenic risk are given including those affecting chemical food additives, consumer product safety, toxic substances, and occupational health and safety. Acceptability of risk is intrinsically a a socioeconomic-political question. While the elements of the exercise may be defined in scientific, objective terms, the content of these terms cannot be reduced to meaningful numbers that h ave more than ephemeral value in the decision-making process. (29 Refs)

GENERAL REVIEWS AND OTHER STUDIES

15. SOME RISK FACTORS OF BREAST CANCER.

Scolozzi R, Salmi R, Coccafogli A, Dell'Acqua G
Istituto di Clinical Medica Generale e Terapia Medica, Universita, Ferrara, Italy
Recenti Prog Med; 70(5):463-486 1981

Studies on the risk factors of breast cancer are reviewed. High stature, overweight (obesity), early menarche, hyperestrogenic, reduced fertility, advanced age at first childbirth, short overall duration of lactation, high-caloric, high-fat diet, long-term use of oral contraceptives, especially before the first pregnancy, late menopause, and exposure to ionizing radiation, especially during and shortly after puberty are generally recognized risk factors along with family history of breast cancer. The long-term use of hair dyes, especially of permanent ones, is also considered a risk factor for breast cancer along with other cancers. (134 Refs) (In ITA)

16. THE PROTECTIVE PROPERTIES OF DIETARY FIBER.

Burkitt DP
The Old House, Bussage, Stroud, Glouster GL6 8AX, England
NC Med J; 42(7):467-471 1981

An increasing consensus that dietary fiber is strongly protective against colon cancer is explored. Several mechanisms which probably act in combination have been postulated to account for this protective action. It is suggested that practical protective measures, such as relatively simple dietary changes, may have highly beneficial effects. (30 Refs)

17. LIFESTYLE AND CANCER.

Higginson J
International Agency for Res. on Cancer, Lyon, France
Cancer Forum; 5(1):4-14 1981

The causation of cancer is discussed with respect to cancers caused by defined exogenous factors and those caused by probable environmental factors. The possible role of general environmental pollution in causing cancer is discussed. Lifestyle is defined as the overall cultural, behavioral, and dietary environments are well as clearly defined habits. The role of lifestyle in the etiology of cancer is discussed with respect to the significance of occupational studies and low risk groups. Clinical cancer should be considered in terms of a summation of events, only a few of which have been identified and studied. The implications of epidemiological observations in relation to the mechanisms of carcinogenesis are reviewed. Epidemiology since 1950 concerning the etiology of cancer is tabulated. Cancer is discussed as a socioeconomic problem. Future research should involve epidemiological studies in order to predict future trends, the differentiation between potential carcinogens which have only been recently discovered and stimuli present in the environment for a significant period, and research on lifestyle factors. (no Refs)

18. FALLACIES AND DISTORTIONS IN THE EPIDEMIOLOGY OF CANCER: PETO VERSUS EPSTEIN AND SWARTZ.

Harington JS
Cancer Res. Dept., Natl. Cancer Assoc. South Africa, P.O. Box 2000, Johannesburg 2000, South Africa
S Afr J Sci; 77(3):108-111 1981

The arguments surrounding Peto versus Epstein and Swartz in relation to the epidemiology of cancer are presented and discussed. According to Epstein, who is supported by Swartz, occupational carcinogens play an important role in the development of cancer. Peto supports the lifestyle attitude which contends that if you get cancer it is due to the way you live, particularly in relation to diet and smoking. These arguments are discussed in relation to cancer deaths in England, Scotland, Wales, and the United States, in South Africa, in Californian Mormons, and in Seventh-day Adventists. In the last two groups, which have highly disciplined lifestyles, the reduction in cancers of all sites are generally impressive (a 37% lower incidence in Mormon males and 33% in females, and about 47% and 32% lower mortality in Seventh-day Adventist males and females, respectively). It was suggested that the arena in which Peto, Epstein, and Swartz are arguing is restricted to 5-10% of all deaths in Western society, that is, those due to lung cancer. It was also suggested that the argument is restricted entirely to the developed countries of England, Wales, and the United States. (15 Refs)

19. COFFEE AND PANCREATIC CANCER: THE PROBLEMS OF ETIOLOGIC SCIENCE AND EPIDEMIOLOGIC CASE-CONTROL RESEARCH.

Feinstein AR, Horwitz RI, Spitzer WO, Battista RN
Robert Wood Johnson Clinical Scholar Program, Yale Univ. Sch. Medicine, 333 Cedar St., Box 3333, New Haven, CT, 06510
JAMA; 246(9):957-961 1981

A commentary is made on the recent report that coffee may cause pancreatic cancer. It contains a review of pertinent principles of case-control methodology, together with a critique of the way these principles were applied in the coffee-pancreatic cancer study to formulate a hypothesis, assemble the case and control groups, collect the individual data, and interpret the results. Arrival at the new hypothesis that coffee may cause pancreatic cancer is likened to results of a fishing expedition; the hypothesis is found to contradict previous findings. The study's apparent lack of attention to elimination of bias in selection and retention of subjects is criticized. The possibility of protopathic bias, in which use of the alleged etiologic agent could have been altered by the same ailment that led to the patient's selection for the study, is considered especially serious. In this connection, the limitedness and lack of verification of data are seen as a major problem. Necessity of statistical allowance for a multiple-contrast experimental situation is stressed: stringent demands on the size of the probability value required for significance are recommended. Doubt about the appropriateness of forming conclusions and extensively diffusing results from a study in which the hypothesis develops as an analytic surprise in the data leads to a plea for re-embracement of scientific experimental principles by case-control investigators. (21 Refs)

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. TOXICOLOGICAL ASPECTS OF POLYCYCLIC AROMATIC HYDROCARBONS (PAH).
De Fernicola NA, De Azevedo FA
Divisao de Toxicologia e Ecotoxicologia, Diretoria de Tecnologia e Desenvolvimento, Sao Paulo, Brazil
Rev Bras Saude Ocup; 33(9):62-68 1981
In POR

2. EVALUATION OF THE CARCINOGENIC RISK OF CHEMICALS TO HUMANS: SOME ANTINEOPLASTIC AND IMMUNOSUPPRESSIVE AGENTS.
International Agency for Research on Cancer
International Agency for Res. on Cancer, Lyon, France
IARC Monogr Eval Carcinog Risk Chem Hum; 26:1-411 1981

3. STUDIES ON THE ROLE OF ORAL CONTRACEPTIVE USE IN THE ETIOLOGY OF BENIGN AND MALIGNANT LIVER TUMORS.
Mettlin C, Natarajan N
Cancer Control and Epidemiology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263
J Surg Oncol; 18(1):73-85 1981

RADIATION CARCINOGENESIS

4. EXPERIMENTAL RADIATION CARCINOGENESIS: WHAT HAVE WE LEARNED?
Fry RJ
Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830
Radiat Res; 87(2):224-239 1981

5. INFLUENCE OF NONCARCINOGENIC SECONDARY FACTORS ON RADIATION CARCINOGENESIS.
Little JB
Lab. Radiobiology, Harvard Univ., Sch. Public Health, Boston, MA, 02115
Radiat Res; 87(2):240-250 1981

6. MEDICAL IRRADIATION IN JAPAN - STOCHASTIC RISK ESTIMATION AND ITS REDUCTION.
Hashizume T
Div. Physics, National Inst. Radiological Sciences, 9-1 Anagawa 4-Chome, Chiba Shi 260, Japan
Nippon Igaku Hoshasen Gakkai Zasshi; 41(5):445-474 1981
In JPN

7. RADIOACTIVITY IN DRINKING WATER.
Cothern CR
Office of Drinking Water, Environmental Protection Agency, Washington, DC
Radioactivity in Drinking Water. Available from National Technical Information Service, Springfield, VA, as PB81-192833:, 79 pp., 1981.

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

8. EPIDEMIOLOGY OF GASTROINTESTINAL CANCER.
Miller AB
NCIC Epidemiology Unit, Univ. Toronto, McMurrich Building, Toronto, Ontario M5S 1A8, Canada
Compr Ther; 7(8):53-58 1981

9. EPIDEMIOLOGICAL ASPECTS OF ESOPHAGEAL CANCER.
Ruggieri M, Tosato F, De Rocco K, Lepore M, Cipriano E, Paolini A
IV Cattedra di Clinica Chirurgica Generale e Terapia Chirurgica, Universita degli Studi di Roma, Rome, Italy
Recenti Prog Med; 70(5):545-556 1981
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10. PANCREATIC CANCER.
Cohn I, Hastings PR
Dept. Surgery, Louisiana State Univ., 1542 Tulane Ave., New Orleans, LA, 70112
UICC Tech Rep Ser. Vol. 57, Issue 12, 142 pp., 1981.

11. GENETICALLY DETERMINED SUSCEPTIBILITY TO CANCER.
Marks PA
Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY, 10021
Blood; 58(3):415-419 1981

12. ETIOLOGY, ROLE AND DETECTION OF CHROMOSOMAL ABERRATIONS IN MAN.
Fabricant JD, Legator MS
Dept. Preventive Medicine, Div. Environmental Toxicology, Univ. Texas Medical Branch, Galveston, TX, 77550
JOM; 23(9):617-625 1981

ENVIRONMENTAL AND OCCUPATIONAL FACTORS IN CARCINOGENESIS

13. INDUSTRIAL CARCINOGENESIS.
Vickers CF
Dept. Dermatology, Univ. Liverpool, Liverpool L69 3BX, England
Br J Dermatol; 105(Suppl21):57-61 1981

14. ENVIRONMENT AND HEALTH (LITERATURE REVIEW).
Khachatrian TS
Dept. Public Health, State Inst. Advanced Training for Physicians, Erevan, USSR
Zh Eksp Klin Med; 21(3):287-292 1981
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15. LIFESTYLE AND CANCER.
Higginson J
International Agency for Res. on Cancer, Lyon, France
Cancer Forum; 5(1):4-14 1981

16. ASBESTOS, AN ENVIRONMENTAL POLLUTANT.
Zamfir G, Diaconu R
Institutul de igiena si sanatate publica, Iasi, Romania
Igiena; 30(1):1-9 1981
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17. REVIEW OF UICC PROGRAM ON SMOKING AND LUNG CANCER.
Gray N
Smoking and Cancer Program, UICC, 3 rue du Conseil General, 1205 Geneva, Switzerland
Cancer Forum; 5(1):15-18 1981

CELLULAR AND MOLECULAR MECHANISMS IN CARCINOGENESIS

18. EXPERIMENTAL APPROACHES TOWARDS THE BIOCHEMICAL ANALYSIS OF CHEMICAL CARCINOGENESIS.
Laishes BA
McArdle Lab. for Cancer Res., Univ. Wisconsin Medical Sch., Madison, WI, 53706
ACS Symp Ser; 160:197-215 1981

19. FIBRONECTIN AND RELATED TRANSFORMATION-SENSITIVE CELL SURFACE PROTEINS (ONCOLOGY OVERVIEW).
International Cancer Research Data Bank
ICRDB Program Office, NCI, Westwood Building, Room 10A18, Bethesda, MD, 20205
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Magrath IT
Pediatric Oncology Branch, Div. Cancer Treatment, NCI, NIH, Public Health Service, United States Dept. Health and Human Services, Bethesda, MD, 20205
JNCI; 67(3):501-514 1981

MODIFICATION OF CARCINOGENESIS

22. FACTORS STIMULATING AND INHIBITING TUMOR GROWTH IN CARCINOGENESIS.
Kochergina NI, Kosova IP
Res. Lab. Experimental Immunobiology, Moscow, USSR
Usp Sovrem Biol; 91(3):409-418 1981
In RUS

23. TWO INDEPENDENT ASPECTS OF TUMOR PROMOTION.
Berenblum I, Armuth V
Weizmann Inst. Science, Rehovot, Israel
Biochim Biophys Acta; 651(1):51-63 1981

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

24. BURKITT'S LYMPHOMA.
Ziegler JL
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N Engl J Med; 305(13):735-745 1981

25. REVIEW ARTICLE. REGULATION OF EXPRESSION OF THE INTEGRATED RETROVIRUS GENOME.
Weinberg RA, Steffen DL
Center for Cancer Res., Massachusetts Inst. Technology, Cambridge, MA, 02139
J Gen Virol; 54(part1):1-8 1981

METHODOLOGY IN CARCINOGENESIS RESEARCH

26. THE DEVELOPMENT AND VALIDATION OF MUTAGENICITY SCREENING TESTS.
Durkin PR, Santodonato J
Center for Chemical Hazard Assessment, Syracuse Res. Corporation, Merill Lane, Syracuse, NY, 13210
Tappi; 64(9):153-159 1981

27. SHORT-TERM TEST SYSTEMS FOR POTENTIAL MUTAGENS AND CARCINOGENS: I. MICROBIAL TESTS (ONCOLOGY OVERVIEW).
International Cancer Research Data Bank
ICRDB Program Office, NCI, Westwood Building, Room 10A18, Bethesda, MD, 20205
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28. ANIMAL MODELS OF INTRA-ORAL CHEMICAL CARCINOGENESIS: A REVIEW.
Eveson JW
Dept. Oral Medicine and Pathology, Guy's Hosp., London SE1 9RT, England
J Oral Pathol; 10(3):129-146 1981

29. THE CHROMOSOMES IN HEMATOLOGY.
Berger R
Laboratoire de Cytogenetique, Institut de Recherches sur les Leucemies et les Maladies du Sang, UER d'Hematologie, Hopital Saint-Louis, 2 place du Dr. Fournier, 75475 Paris Cedex 10, France
Cancer Genet Cytogenet; 4(1):69-88 1981

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DIETARY ASPECTS OF CARCINOGENESIS

SERIES CK03 No. 82/02

FEBRUARY, 1982

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In this issue:

- STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET 1-2
- STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET 3-4
- EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET 5-6
- MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS 7-8
- GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS 9-13
- GENERAL REVIEWS AND OTHER STUDIES 14-15

STUDIES OF NATURALLY OCCURRING

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • National Institutes of Health • National Cancer Institute

HE 20. 3173/2:

CARCINOGENS IN THE DIET

1. COMPARISON OF THE MUTAGENICITY OF THE DIET IN RELATION TO MORTALITY FROM GASTRIC CANCER. I. THE FOODS EATEN IN NARA AND KAGOSHIMA FROM THE VIEWPOINT OF THE MUTAGENICITY OF THE FOODS EATEN IN AKITA AND IWATE (MEETING ABSTRACT).

Kamiyama S, Michioka I
Dept. Hygiene, Akita Univ., Sch. Medicine, Hondo, Akita 010, Japan
Nippon Eiseigaku Zasshi; 36(1):177 1981

The mutagenicity of the foods of the inhabitants of the farm and mountain villages of Nara and Kagoshima which have contrasting mortality from gastric cancer was examined and compared with that of the inhabitants of Akita and Iwate, reported earlier. The diet of the head or housewife of the family of 30 and 31 households, respectively, in Haibara-machi, Nara Prefecture, and Togo-cho, Kagos hima Prefecture, selected randomly in May 1980 was recorded for 2 days. The foods which were eaten significantly more in a mutagenic diet and those which were eaten significantly more in a nonmutagenic diet were referred to as positive and negative foods, respectively. The percentage of positive foods eaten in Nara and Kagoshima was similar to that in Akita and Iwate, respectively. On the other hand, the percentage of negative foods was the highest in Kagoshima, followed in order by Nara, Iwate and Akita. The number of negative foods was plotted against that of the positive foods to obtain a stochastic ellipse. There was a clear difference between the number of positive and negative foods in a positive diet and that in a negative diet. The mortality for gastric cancer between 1973 and 1977 was 68.05, 38.56, 27.27 and 11.03 per 100,000 for Nara, Akita, Iwate and Kagoshima, respectively. The number of the negative and positive foods consumed was clearly reflected in these mortalities. (no Refs) (In JPN)

2. COMPARISON OF THE MUTAGENICITY OF THE DIET IN RELATION TO MORTALITY FROM GASTRIC CANCER. II. MUTAGENICITY OF THE FOODS INGESTED BY THE INHABITANTS OF THE COUNTRYSIDE OF NARA AND KAGOSHIMA (MEETING ABSTRACT).

Michioka I, Kamiyama S
Dept. Hygiene, Akita Univ., Sch. Medicine, Hondo, Akita 010, Japan
Nippon Eiseigaku Zasshi; 36(1):178 1981

Foods to be consumed by the inhabitants of the villages in Haibara-machi, Nara Prefecture, and Togo-cho, Kagoshima Prefecture, which have different mortalities from gastric cancer, were frozen and air-mailed to this facility where they were thawed and the mutagenicity of the acetone-soluble fraction of the ether:petroleum ether (1:1) extract of the homogenate of the edible part was determined by the Ames' method with Salmonella typhimurium TA100. The foods with a relative mutagenicity rate of less than 1.5, 1.5-3.0 and more than 3.0 in the extract were designated negative, false-positive and positive, respectively. The rate of ingestion of negative and positive foods was strongly related to the mutagenicity of the foods for Akita and Iwate. However, in Nara the positive mutagenicity rate was low compared to that of gastric cancer mortality, which was high. The presence of some factors which promote the action of the mutagenic substances in foods on the gastric epithelium was thought to be responsible for the incidence of gastric cancer in Nara. (no Refs) (In JPN)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

3. THE CARCINOGENICITY OF ARSENIC.

Pershagen G
Dept. Environmental Hygeine, Karolinska Inst., Stockholm, Sweden
Environ Health Perspect; 40:93-100 1981

Assessment of arsenic (As) as a carcinogen is reviewed. As occurs widely in the environment, being found in various ores, coal, tobacco, pesticides, water, air, and foodstuffs such as marine fish and crustaceans, swine and poultry, as well as in industrial processes. Distinguishing between organic and inorganic As is important in determining carcinogenicity. Only inorganic As has so far been linked to cancer. Epidemiological studies have associated lung cancer with occupational exposure to As in pesticide workers, copper smelters, and vintners using pesticides. Skin cancer has been associated with exposure to As in drinking water in several reports. Neoplastic changes in the skin include Bowen's disease and basal and squamous cell carcinomas. Experimental studies in animals have not provided conclusive evidence of AsS carcinogenicity. Investigation of metabolism indicates that As may interfere with DNA repair mechanisms. Recommendations for future research are made. (101 Refs)

4. HEALTH RISKS FROM VOLATILE HALOGENATED HYDROCARBONS?

Lahl U, Cetinkaya M, VDuszeln J, Stachel B, Thiemann W
Dept. Biology/Chemistry, Univ. Bremen, Leobener Str., D-2800 Bremen 33, W. Germany
Sci Total Environ; 20(2):171-189 1981

Human exposure to volatile halogenated hydrocarbons (VHH) and associated risks to health are reviewed. VHH are found in drinking water, air, and food. In West Germany the daily av burden for the most important VHH is in the range 0.1-1 ug/kg/day. However, many additional burdens of VHH which are not included in mean values of body-burden include occupational exposure, contaminated ground water, ingestion of contaminated fish, disinfection sol residue on infant feeding utensils, and ingestion of chloroform in swimming pools. Epidemiologic data reveal the carcinogenicity of VHH in man. Animal studies and in vitro studies support the hypothesis that VHH are carcinogenic and mutagenic in animals. Extrapolation of experimental findings to man and extrapolation from the necessary high doses used in bioassay to the substantially lower quantities found in the environment are discussed. The use of VHH in open systems in industry, food production, households, etc, should be minimized by the use of appropriate substitutes, better and stricter regulations, and appropriate control. (62 Refs)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

5. A POTENT MUTAGEN IN BROILED FISH. PART 1. 2-AMINO-3-METHYL-3H-IMIDAZO[4,5-F]QUINOLINE.

Kasai H, Yamaizumi Z, Nishimura S, Wakabayashi K, Nagao M, Sugimura T, Spingarn NE, Weisburger JH, Yokoyama S, Miyazawa T
Biology Div., Natl. Cancer Center Res. Inst., Chuo-ku, Tokyo, Japan
J Chem Soc [Perkin I]; (8):2290-2293 1981

Sun-dried sardines were broiled in normal fashion, ground, and extracted with methanol. The residue showed strong mutagenicity in Salmonella typhimurium strain TA98 in the presence of S-9 mixture, according to the Ames method. After further fractionation a neutral fraction was obtained that re tained 40% of the mutagenicity of the original fraction. Purification yielded a mutagenic compound. The compound was identified by nuclear magnetic resonance spectrum and high- and low-resolution mass spectra as 2-amino-3-methyl-3H-imidazo[4,5-f]quinoline (AMIQ). The identification was confirmed by synthesis of the compound. The structure is a six-six-five-membered ring. The mutagenicity of AMIQ was much higher than that of other mutagens isolated from protein pyrolysis products. The cause of the high mutagenicity is as yet unknown. (32 Refs)

6. EFFECTS OF PHENOBARBITAL PRETREATMENT ON THE METABOLISM AND TOXICOKINETICS OF AFLATOXIN B1 IN THE RHESUS MONKEY.

Wong ZA, Wei CI, Rice DW, Hsieh DP
(c/o Hsieh) Dept. Environmental Toxicology, Univ. California, Davis, CA, 95616
Toxicol Appl Pharmacol; 60(3):387-397 1981

Five male rhesus monkeys were treated with phenobarbital (PB: 15 mg/kg, 2 x/day) for 1 wk prior to administration of aflatoxin B1 (AFB1: 0.1-1.0 mg/kg, iv). PB pretreatment enhanced the in vitro conversion of AFB1 to aflatoxin Q1 and aqueous metabolites in monkey liver. Although PB did not appreciably alter the rate or vol of AFB1 distribution in vivo, it reduced plasma levels of total aflatoxins, AFB1, and the toxic metabolite aflatoxin M1 (AFM1). Decreased AFM1 levels and increased levels of less toxic water-soluble metabolites were found in the urine of PB-treated monkeys. These findings demonstrated that increased hepatic AFB1 metabolism results in elimination and circulation of less toxic, less mutagenic, and highly excretable metabolites. (49 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

7. A NOTE ON THE ROLE OF DIETARY RETINOL AND CAROTENE IN HUMAN GASTRO-INTESTINAL CANCER.

Modan B, Cuckle H, Lubin F
Dept. Clinical Epidemiology, Chaim Sheba Medical Center, Tel Hashomer and Tel Aviv Univ. Medical Sch., Tel Aviv, Israel
Int J Cancer; 28(4):421-424 1981

Dietary data relating to 406 patients with gastrointestinal cancers (cases) and 812 controls have been analyzed to test the hypotheses that dietary vitamin A (retinol) and its precursor, carotene, may be protective agents in human carcinogenesis. There was no deficit among cases in the intake of retinol-containing food items whereas several of the main carotene-containing fruits and vegetables were eaten less often among cases than among controls. However, when total daily intake was estimated, there was no protective effect of carotene, perhaps implying that some other constituent of the fruits and vegetables is protective. (Author abstract) (11 Refs)

8. DIETARY SELENIUM INTAKE AND GROWTH OF THE MT-W9B TRANSPLANTABLE RAT MAMMARY TUMOR.

Ip C, Ip MM, Kim U
Dept. Breast Surgery, Roswell Park Memorial Inst., Buffalo, NY, 14263
Cancer Lett; 14(1):101-107 1981

The effect of dietary selenium deficiency and supplementation on the growth of the transplantable MT-W9B mammary carcinoma in syngeneic Wistar-Furth female rats was investigated. The rats were fed a high fat diet (25% corn oil) supplemented with either less than 0.02 ppm, 0.1 ppm, or 2 ppm selenium. Selenium deficiency caused a drastic reduction in the activity of hepatic glutathione peroxidase, while excess selenium had little effect on its activity. Palpable tumors appeared earlier in the rats given less than 0.02 ppm or 0.1 ppm selenium, compared to those given 2 ppm. Dietary selenium at 2 ppm reduced the tumor wt by approx 50% compared to that in the 0.1 ppm group. No histological change in the tumors accompanied with the wt decrease. It is suggested that the inhibitory effect of selenium is selective, without inducing any wt loss or any recognizable toxicity in the animals. (25 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

9. HUMAN SALIVA INACTIVATES MUTAGENICITY OF CARCINOGENS.

Nishioka H, Nishi K, Kyokane K
Div. Biochemistry, Doshisha Univ., Kyoto, Japan
Mutat Res; 85(5):323-333 1981

Various carcinogens, suspected carcinogens, certain pyrolysates (beef, salmon, sodium glutamate, polypeptone), and cigarette smoke condensate (CSC) were incubated with human saliva from healthy subjects. Effects on mutagenicity were tested in a modified Ames test with Salmonella typhimurium strains TA 98 and TA 100. Metabolic activation for development of mutagenicity was required by aflatoxin B1, benzo(a)pyrene (BP), tryptophan pyrolysate-1 (Tr-P-1, isolated from boiled beef), the other pyrolysates, and CSC. No metabolic activation was required by 2-(2-furyl)-3-(5-nitro-2-furyl)acrylamide (AF-2; a nitrofuran used as a food additive during 1966-1975 in Japan), N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), methyl methanesulfonate (MMS), 4-nitroquinoline 1-oxide (4NQO) and quercetin. Saliva inactivated AF-2, MNNG, 4NQO, aflatoxin B1, BP, and Trp-P-1. Mutagenic activities of quercetin, pyrolysates of beef, salmon, and sodium glutamate, and CSC decreased to some extent; the activities of MMS and polypeptone pyrolysate were not affected. Boiled saliva inactivated AF-2 somewhat in TA 98 but not in TA 100 cultures. Complex mechanisms may be involved in the inactivation of mutagenicity of carcinogens by saliva. (22 Refs)

10. CANCER OF THE COLON AND RECTUM IN THE COLOURED POPULATION OF JOHANNESBURG. RELATIONSHIP TO DIET AND BOWEL HABITS.

Maisto OE, Bremner CG
(c/o Bremner) Dept. Surgery, Univ. Witwatersrand Medical Sch., Esselen St., Hillbrow, Johannesburg, 2001, Republic of South Africa
S Afr Med J; 60(15):571-573 1981

In the Colored population of Johannesburg, the incidence of carcinoma of the colon and rectum estimated over an 11-yr period (1970-1980) is 133 and 108/100,000 respectively per yr. This is lower than the incidence reported in Whites and appears to be even lower than that reported in South African and Zimbawean Blacks. A survey of dietary factors in this population showed a diet which was high in protein, low in fats and high in bulk. The frequency of bowel action was higher than in both Whites and Blacks. These factors support Burkitt's hypothesis which states that races with a high-bulk diet have a lower incidence of large-bowel cancer. (Author abstract) (16 Refs)

11. ESOPHAGEAL CANCER AMONG BLACK MEN IN WASHINGTON, D.C. I. ALCOHOL, TOBACCO, AND OTHER RISK FACTORS.

Pottern LM, Morris LE, Blot WJ, Ziegler RG, Fraumeni JF
Environmental Epidemiology Branch, Div. Cancer Cause and Prevention, NCI, NIH, Public Health Service, U.S. Dept. Health and Human Services, Bethesda, MD, 20205
JNCI; 67(4):777-783 1981

A case-control study involving interviews with the next of kin or close friends of 120 black males who recently died of esophageal cancer and 250 similarly aged black males who died of other causes was undertaken to discover reasons for the exceptionally high mortality from this cancer in Washington, DC. The age-adjusted annual death rate in Washington, DC, for nonwhite males, 1970-75, was 28.6/100,000 far higher than the national rate of 12.4/100,000 and the rates in other metropolitan areas of the country. The major factor responsible for the excess was alcoholic beverage consumption, with an estimated 81% of the esophageal cancers attributed to its use; high use of alcoholic beverages was also found among the controls. The relative risk (RR) of esophageal cancer associated with use of alcoholic beverages was 6.4 (95% confidence interval = 2.5, 16.4). The RR increased with amount of ethanol consumed and was highest among drinkers of hard liquor, although the risk was also elevated among consumers of wine and/or beer only. The RR associated with cigarette smoking was 1.9 (1.0, 3.5) when controls with smoking-related causes of death were excluded but declined to 1.5 (0.7, 3.0) when adjusted for ethanol consumption. Significant differences of approx twofold were found between low and high levels of (a) consumption of fresh or frozen meat and fish, fruits and vegetables, and dairy products and eggs and (b) relative wt (wt/ht2). The inverse trends with these general measures of nutritional status were not explained by alcoholic beverage consumption of socioeconomic status as measured by educational level. (Author abstract) (23 Refs)

12. CARCINOGENIC FACTORS IN MALIGNANT TUMORS OF THE HEAD AND NECK (PART 2) - INVESTIGATION OF ALCOHOLISM IN PATIENTS.

Miyahara H, Sato T
Dept. Head and Neck Surgery, Center for Adult Diseases, 1-3-3 Nakamichi, Higashinari-ku, Osaka 537, Japan
Nippon Jibiinkoka Gakkai Kaiho; 84(2):145-153 1981

Twelve male patients, 49-75 yr old, who were alcoholics and had malignant tumors of the head and neck were studied. The tumors were in the mesopharynx in five patients, in the oral cavity in four, in the larynx in three, in the esophagus in two, in the hypopharynx in one, and in the parotid gland in one; four of the patients were thought to have multiple tumors. Eight of the patients had Stage III tumors, and one had a Stage IV tumor. Of the 12 patients, 8 died of cancer within 1 yr of treatment. All patients smoked tobacco, and consumption of tobacco was assessed by Brinkman's index: the lowest value was 400, and the highest was 2,200. With respect to alcohol consumption, patients from the Kumamoto area primarily drank shochu, with the lowest consumption being greater than 3.6 liters/day for greater than 20 yr. Four patients had been repeatedly admitted to the hospital due to alcohol poisoning. One of the patients with lower pharyngeal and esophageal cancer drank 0.5-1 bottle of whiskey/day and smoked 100 cigarettes/day. Complications associated with alcoholic damage included gastritis, gastric ulcers, hepatitis, hepatic cirrhosis, diabetes, and hypertension. Nine patients showed no skin response to dinitrochlorobenzene. Results suggested that heavy drinking and smoking are carcinogenic. (8 Refs) (In JPN)

13. EVALUATION OF POSSIBLE CAUSES OF DELAYED HYPERSENSITIVITY IMPAIRMENT IN CANCER PATIENTS.

Dominioni L, Dionigi R, Dionigi P, Nazari S, Fossati GS, Prati U, Tibaldeschi C, Pavesi F
Istituto de Patologia Chirurgica, Universita de Pavia, 27100 Pavia, Italy
JPEN; 5(4):300-306 1981

The role of tumor growth, malnutrition, and aging as etiologic factors of depressed delayed hypersensitivity response (DHR) was studied in patients (48 men, 63 women; 20-80 yr old) with solid tumors (20 stomach, 16 colon, 18 rectum, 27 breast, and 30 other cancers). The sex distribution, mean age, and mean serum albumin (SA) levels of the cancer patients and 56 controls (27 men, 29 women; 30-88 yr old) were not significantly different. The controls consisted of nonneoplastic patients, many of whom presented with clinical signs of overt malnutrition, who had been referred to the institute because of their poor general condition and high surgical risk. The mean SA levels of both these cancer patients and controls were significantly lower than the mean serum albumin level of well-nourished, age-matched controls. Significantly depressed values of SA and DHR were recorded in patients with stomach, colon, and rectum cancers. The stage of the tumor was inversely correlated with the SA level. Cancer patients presented a mean DHR that was not significantly different from the DHR of matched controls; however, the DHR of both groups were significantly lower than that of hospitalized patients, less than 50 yr old with minimal nonneoplastic diseases. All anergic cancer patients, except one, had tumors of the gastrointestinal tract and a very low me an SA level. The mean tumor stage of anergic cancer patients was significantly more advanced than in the remaining anergic cancer patients. Regression analysis indicated a significant correlation between the DHR and SA indicating that solid tumors cause malnutrition and that, subsequently, malnutrition causes depression of DHR. (57 Refs)

GENERAL REVIEWS AND OTHER STUDIES

14. NUTRITIONAL MODULATION OF CANCER.

Nauss KM, Newberne PM
Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA, 02139
Cancer Detect Prev; 3(2):367-417 1981

A review is made of (1) specific target organs where epidemiologic and experimental animal data indicate a role for nutrition in tumor development, (2) the role of certain vitamins, especially vitamin A and ascorbic acid, in modulation of cancer, and (3) experimental evidence for a nutritional role in the modification of carcinogen metabolism and the host immune response. Nutritional deficiencies are etiologic factors in esophageal and gastric cancer, malnutrition and dietary carcinogens etiologic factors in liver cancer, and dietary fats etiologic factors in colon cancer. Dietary components that inhibit the metabolic activation of carcinogens include flavones, indoles, and antioxidants. Vitamin A and its synthetic analogs are effective inhibitors of carcinogen-induced epithelial neoplasms; they also enhance cellular immunity and antitumor response. (140 Refs)

15. CARCINOGENESIS OF GASTRIC CANCER WITH SPECIAL REFERENCE TO ETIOLOGICAL ASPECTS.

Schmahl D
Deutsches Krebsforschungszentrum, Postfach 101 949, D-6900 Heidelberg 1, W. Germany
Zentralbl Chir; 106(15):997-1004 1981

The article discusses possible etiological factors for the carcinogenesis of stomach tumors. Apart from genetic factors, stomach carcinogenesis may be attributed to the exposure of the stomach to carcinogenic substances taken in with the food, such as pyrolysis products of certain amino acids, N-nitroso compounds or disturbances of the physiological function of the stomach, for example pathological reflux after stomach operations. The frequency of stomach cancer could not be related to occupational exposure. Geographic pathology has shown that people coming from high risk areas (eg Japan) adapt to the risk pattern of the host country when they emigrate (eg to the United States, where there is only a low risk for stomach cancer). Therefore, it is assumed that several factors play a role in the etiology of stomach cancer. (Author abstract) (16 Refs) (In GER)

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. CHEMICAL COMPOUNDS AND CANCER RISK.

Schramm T, Janisch W
Zentralinstitut fur Krebsforschung der Akademie der Wissenschaften der DDR, Lindenberger Weg 80, DDR-1115 Berlin, E. Germany
Arch Geschwulstforsch; 51(7):561-565 1981
In GER

2. THE CARCINOGENICITY OF ARSENIC.

Pershagen G
Dept. Environmental Hygiene, Karolinska Inst., Stockholm, Sweden
Environ Health Perspect; 40:93-100 1981

3. THE CARCINOGENICITY OF CHROMIUM.

Norseth T
Inst. Occupational Health, Gydas vei 8, Oslo 3, Norway
Environ Health Perspect; 40:121-130 1981

4. ROLE OF COBALT, IRON, LEAD, MANGANESE, MERCURY, PLATINUM, SELENIUM, AND TITANIUM IN CARCINOGENESIS.

Kazantzis G
TUC Centenary Inst. Occupational Health, London Sch. Hygiene and Tropical Medicine, Keppel St., London WC1E 7HT, England
Environ Health Perspect; 40:143-161 1981

5. RECENT RESEARCH ON NICKEL CARCINOGENESIS.

Sunderman FW
Dept. Lab. Medicine, Univ. Connecticut Sch. Medicine, Farmington, CT, 06032
Environ Health Perspect; 40:131-141 1981

6. ROLE OF CADMIUM IN CARCINOGENESIS WITH SPECIAL REFERENCE TO CANCER OF THE PROSTATE.

Piscator M
Dept. Environmental Hygiene, Karolinska Inst., S-104 01 Stockholm, Sweden
Environ Health Perspect; 40:107-120 1981

7. CHELATING AGENTS IN BIOLOGICAL SYSTEMS.

Schubert J
Dept. Chemistry, Univ. Maryland Baltimore County, Catonsville, MD, 21228
Environ Health Perspect; 40:227-232 1981

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Dept. Nutrition and Food Science, Massachusetts Inst Technology, Cambridge, MA, 02139
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Cancer Res. Dept., Natl. Cancer Assoc. South Africa, P.O. Box 2000, Johannesburg 2000, South Africa
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Grossman L
Dept. Biochemistry, Sch. Hygiene and Public Health, Johns Hopkins Univ., 615 N. Wolfe St., Baltimore, MD, 21205
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Zakour RA, Kunkel TA, Loeb LA
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Vainio H, Sorsa M
Dept. Industrial Hygiene and Toxicology, Inst. Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland
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Nordberg GF, Andersen O
Dept. Environmental Hygiene, Umea Univ., Umea, Sweden
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Inst. Environmental Medicine, New York Univ. Sch. Medicine, 550 First Ave., New York, NY, 10016
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Latt SA, Allen J, Bloom SE, Carrano A, Falke E, Kram D, Schneider E, Schreck R, Tice R, Whitfield B, Wolff S
Harvard Medical Sch., Boston, MA, 02115
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Sirover MA
Dept. Pharmacology, Temple Univ. Sch. Medicine, Philadelphia, PA, 19140
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STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. COFFEE AND TEA CONSUMPTION AND BREAST DISEASE.

Lawson DH, Jick H, Rothman KJ
(c/o Jick), Boston Collaborative Drug Surveillance Program, 400-1 Totten Pond Rd., Waltham, MA, 02154
Surgery; 90(5):801-803 1981

The relation of coffee and tea consumption to fibrocystic disease of the breast and to breast cancer was studied in 451 women hospitalized for these diseases and interviewed since 1972. We found a modest, positive association between hot beverage consumption and both diseases. However, there was

HE 20.3173/2:

no apparent dose-response effect. These data add little support to the hypothesis that methylxanthine cons umption plays an etiologic role in the development of fibrocystic or malignant disease of the breast. (Author abstract) (5 Refs)

2. POTENCY OF AFLATOXIN B1 AND AFLATOXIN M1 IN CYTOTOXICITY AND DNA REPAIR ASSAYS (MEETING ABSTRACT).

Green CE, Rice DW, Hsieh DP, Byard JL

Department of Environmental Toxicology, University of California, Davis, CA, 95616

Toxicologist; 1(1):42 1981

Although the acute toxicity and carcinogenicity of aflatoxin B1 (AFB1) has been thoroughly documented, relatively little is known of the potency of aflatoxin M1 (AFM1), a hydroxylated metabolite of AFB1. Therefore, the cytotoxicity and genotoxicity of AFB1 and AFM1 were compared using in vitro system of primary cultures of rat hepatocytes. Hepatocytes were cultured for 20 hr (2.5 x 10(6) cells/dish) in hormone supplemented culture medium. The incubation was continued for an additional 24 hr with the test chemicals and 0.5 uCi/ml [3H] thymidine ([3H]TdR). Cytotoxicity was assessed by measuring lactate dehydrogenase released into the culture medium. Both AFB1 and AFM1 were potent cytotoxins. LDH levels were elevated significantly above controls for AFB1 at 0.1 ug/dish and for AFM1 at 0.6 ug/dish. However, LDH concentrations rose to the same max of approx 200% of controls for AFB1 and AFM1 at a dose of 1.2 ug/dish. Genotoxicity was assessed by measuring unscheduled DNA synthesis by two different methods, incorporation of [3H] TdR into trypsin-resistant acid-precipitable material and autoradiographic analysis. AFB1 and AFM1 were both positive and about equally potent in the DNA repair assay using either technique. In vivo experients indicate a higher level of DNA binding for AFb1 than AFM1. There are two plausible explanations for the DNA repair data; 1) AFB1 and AFM1 bind to DNA to approx the same extent in the hepatocyte culture system, 2) less AFM1 is covalently bound to DNA than AFB1 but AFM1 adducts are more efficiently repaired. (1 Ref)

3. LIVER CANCER IN RAINBOW TROUT: ITS SUDDEN APPEARANCE AND DISAPPEARANCE IN WEST GERMANY DURING THE PERIOD 1968-1973. ITS CAUSES AND CONTROL MEASURES.

Wunder W

I. Zoologisches Institut, Universitat Erlangen-Nurnberg, Universitatsstrasse 19, D-8520 Erlangen, W. Germany

Med Welt; 32(41):1548-1551 1981

Hepatoma was observed in rainbow trouts in West Germany due to the use of dry feed containing aflatoxin-contaminated oil seeds. Sexually immature fish were more sensitive: 60-80% of such fish developed hepatoma. Sexually mature male became practically resistant to aflatoxins, and adult females were also less sensitive than fingerlings, as only up to 5% of adult females were affected. After reaching a size of approx 7 cm, the tumors underwent cystic degeneration, and tumor cells were not demonstrable. The epidemic disappeared after feed free from aflatoxins was introduced. (25 Refs) (In GER)

4. AFLATOXIN CARRYOVER FROM THE DIET AND TISSUE CLEARANCE IN THE PIG (MEETING ABSTRACT).

Furtado RM, Pearson AM, Hogberg MG, Miller ER, Gray JI, Aust SD

Departments of Food Science and Human Nutrition, Michigan State University, East Lansing, MI, 48824

Toxicologist; 1(1):33 1981

Two feeding trials with 20 pigs in each were conducted to ascertain the length of time required to obtain tissue clearance after being fed 42 days on a diet spiked with 551 and 355 ug of aflatoxins B1 and B2/kg feed, respectively. In trial 1, the basal diet contained 20 and 31 ug of aflatoxins B1 and B2/kg feed, respectively, being due to naturally contaminated corn. Contrary to earlier work and results from trial 2, there was no significant difference in wt gains or liver wt between pigs fed the basal and spiked diets. In both trials, low levels of aflatoxins were in the tissues at the end of the feeding trial, mostly in the kidneys and livers as aflatoxins M1 and M2. After 48 hr withdrawal, 7 out of 8 animals were free of aflatoxins, with one pig still showing traces. All pigs were free of measurable aflatoxins after a 4 day withdrawal period. Results indicated that the pig readily metabolized aflatoxins after withdrawal from the diet. Cooking and processing were shown to have little effect upon aflatoxin levels in either fresh or cured pork. Furthermore, dripping collected on cooking were virtually devoid of aflatoxins, confirming the fact that they are largely confined to the water soluble fraction of the tissues. Cooking and/or curing of cuts from meat containing aflatoxins had little or no effect upon residual levels. (no Refs)

5. THE NATURE OF THE RESISTANCE TO CYTOTOXICITY INDUCED IN HEPATOCYTES BY FEEDING A CARCINOGENIC REGIME OF AFLATOXIN B1 TO MALE FISCHER RATS (MEETING ABSTRACT).

Neal GE, Cabral JR

Toxicology Unit, Medical Research Council Laboratories, Wordmansterne Road, Carshalton, Surrey, SM5 4EF, England

Toxicologist; 1(1):34 1981

Evidence is emerging that in the case of hepatocarcinogenesis by low-level administration of many different carcinogens, the irreversible carcinogenic change takes place at a time when the level of presumptive mutagenic lesions in the hepatic DNA is very much lower than that present following subcarcinogenic dosing. Aflatoxin B1 action clearly demonstrates this effect. It has been suggested that the mechanism is an enhanced repair of mutagenic lesions, but the kinetics of DNA binding of labelled aflatoxin are incompatible with this theory. Reduced binding of aflatoxin appears prinicipally to be due to enhanced detoxification of the activated toxin, and this has been demonstrated in vitro binding experiments. Induction of high levels of reduced glutathione and probably S-transferase enzymes appears to be responsible for the effect. These changes are principally, if not exclusively, located in foci of gamma glutamyl transferase positive cells. Evidence for the significance of these changes in terms of induction of preneoplastic lesions will be presented. (no Refs)

6. INHIBITION OF RAT LIVER AMINOACYL-TRNA SYNTHETASES IN VITRO AFTER ACUTE AND CHRONIC AFLATOXIN B1 ADMINISTRATION IN VIVO.

Wagner G, Unterreiner AM
Institut fur Physiologische Chemie I der Universitat, Moorenstr. 5, D-4000 Dusseldorf, W. Germany
Chem Biol Interact; 37(1):233-244 1981

In a cell-free system the influence of acute aflatoxin B1 administration on the fractions of the hepatic microsomal translation complex of female rats was examined and compared with the influence of chronic aflatoxin B1 treatment. Polypeptide synthesis determined by [14C]leucine incorporation with a postmitochondrial supernatant was inhibited by 80% 24 hr after acute aflatoxin B1 administration whereas inhibition was only 17% in animals 30 wk after chronic treatment. After acute aflatoxin B1 administration inhibition of protein synthesis was by 67%, mainly on the polysomal level, whereas inhibition with the pH 5 enzyme was only by 22%. After chronic aflatoxin B1 administration inhibition was by 18%, mainly with the pH 5 enzyme and by only 6% on the polysomes. The inhibition by pH 5 enzyme was further investigated with regard to aminoacyl-tRNA synthetases, components of pH 5 enzyme. Leucine-specific aminoacyl-tRNA synthesis was inhibited by 20% 24 hr after acute and by 12% 30 wk after chronic aflatoxin B1 treatment. Aminoacyl-tRNA synthetase activities in a crude synthetase preparation tested with five amino acids were found to be noncompetitively inhibited. It is concluded that the inhibition of protein synthesis after acute and chronic aflatoxin B1 administration is in part due to decreased aminoacyl-tRNA levels caused by inhibited aminoacyl-tRNA synthetases. (Author abstract) (39 Refs)

7. METABOLISM OF CYCLOPROPENOID FATTY ACIDS AND AFLATOXIN B1 IN ISOLATED HEPATOCYTES OF RAINBOW TROUT (SALMO GAIRDNERI) (MEETING ABSTRACT).

Taylor MJ, Bailey GS, Selivonchick DP
Department of Food Science, Oregon State University, Corvallis, OR 97331
Toxicologist; 1(1):34 1981

Rainbow trout are the most sensitive animal to Aflatoxin B1 (AFB) hepatocarcinogenicity. AFB1 is a potent carcinogenic mycotoxin occurring in contaminated peanuts, corn, and other grains. Trout feeding studies have also demonstrated the great synergistic hepatocarcinogenicity between AFB1 and cyclopropenoid fatty acids (CPFA). CPFA occur naturally in cotton-seed, kapok, and other food oils. Reason for this synergism has been the subject of much research. Hepatocytes were isolated in high viability (90% lactic dehydrogenase retained) from rainbow trout liver in situ by a two-step collagenase perfusion. Cells were incubated with CPFA and AFB1 and various metabolic parameters assessed. CPFA caused a dose-dependent fall in cellular glutathione (GSH) level, and a concomitant rise in lipid peroxidation occurred even in the absence of added ADP-Fe. The drop in GSH content was slower with CPFA than with diethyl maleate (DEM). Great excesses of either CPFA or DEM were required to significantly decrease cellular GSH. Binding of 3H-AFB1 to DNA was also investigated. AFB1 was rapidly bound to DNA in isolated hepatocytes. A dose of AFB1 added to cells after 0,1,2,3 or 4 hr preincubation resulted in the same level of DNA binding. Benzo(a)pyrene monooxygenase activity also remained constant for 4 hr. Cytotoxicity of AFB1 and the effect of CPFA on AFB1-DNA binding will be presented. (no Refs)

8. BLOOD ALPHA-FETOPROTEIN CHANGES IN RATS FED AFLATOXIN B1 AND VARIOUS LEVELS OF CABBAGE.

Boyd JN, Stoewsand GS
Dept. Food Science and Technology, Cornell Univ., New York State Agricultural Experiment Station, Geneva, NY, 14456
J Food Sci; 46(6):1923-1926 1981

Weanling male Fischer rats were fed semi-purified diets containing 0, 5, 10, 20 or 30% freeze-dried ground cabbage for 11 wk. 'Control' groups received no aflatoxin B1 (AB1), 'Ad lib AB1' groups received 1 ppm AB1 in the diets, and 'Meal-fed AB1' groups received two weekly 'meals' of AB1 in 3-5g of purified diet. The amounts of AB1 consumed per wk were the same for both AB1 treated groups. Blood levels of alpha-fetoprotein (AFP), an onco-developmental gene product, were in the order: 'Meal-fed AB1' greater than 'Ad-lib AB1' greater than 'Control'. Inhibition of AFP elevation by cabbage diets in both AB1-treated groups suggested that this vegetable may produce a hepatocarcinogenic protective effect. This effect has been observed in rats fed cauliflower. Inhibition of AFP induction was correlated with increasing levels of dietary cabbage up to 20%. Cabbage diets also resulted in decreased intestinal transmit time and increased relative liver wt. (Author abstract) (25 Refs)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

9. THE TWO-GENERATION SACCHARIN BIOASSAYS (MEETING ABSTRACT).

Arnold DL
Health Protection Branch, Ottawa, Canada
International Symposium on Health Effects of Tumor Promotion, October 12-15, 1981, Cincinnati, Ohio US Environmental Protection Agency and the American College of Toxicology; 1981

Saccharin was discovered over 100 yr ago and the controversy regarding its safety for human consumption started shortly therafter and has not subsided appreciably in the intervening decade. The consumption of saccharin, particulary in North America, started to escalate when under new standards of identity, the US Food and Drug Administration allowed foods containing artificial sweeteners to be promoted as 'nonnutritive' or 'noncaloric' sweeteners for use by the general public. In 1969, when cyclamates were banned, at least 10 single generation feeding studies were undertaken to more accurately assess the toxicological consequences resulting from the anticipated increased consumption of saccharin. While none of these studies resulted in any overt regulatory action, the introduction of the two-generation chronic toxicity/carcinogenicity bioassay added a new total to the toxicologists' arsenal. Three studies have since been conducted using the protocol, and the results from these studies clearly show that when rats were exposed to diets containing 5 or 7.5% sodium saccharin from the time of conception to death, and increased frequency of urinary bladder cancers were predominately found in the males. While some study results suggested that impurities in commercial saccharin, or the presence of urinary tract calculi may have been responsible for the observed bladder tumors, it now appears that these possibilities are highly unlikely. The mechanisms by which saccharin elicited the bladder tumors using the two-generation experimental model has not been ascertained. (no Refs)

10. NITRITE STABILITY AND NITROSATION POTENTIAL IN HUMAN GASTRIC JUICE.

Tannenbaum SR, Moran D, Falchuk KR, Correa P, Cuello C
Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA, 02139
Cancer Lett; 14(2):131-136 1981

Human gastric juice from patients free of chronic atrophic gastritis (CAG) and intestinal metaplasia (IM) was shown to contain factors which rapidly destroy nitrite. Continued generation of nitrite as a result of hypochlorhydria was found to deplete gastric juice of its protective factors and lead to an elevated rate of nitrosation. It is suggested that ascorbic acid and alpha-tocopherol may be useful in areas where gastric cancer is prevalent since these drugs destroy nitrite. (17 Refs)

11. ADVISING PATIENTS ABOUT NITRITES IN FOODS.

Newell GR, Boutwell WB
M. D. Anderson Hosp., Houston, TX
Cancer Bull; 33(4):180-181 1981

Dietary nitrites as additives, natural products, and carcinogens are discussed. Studies relating nitrites to cancer in experimental animals are described. During risk/ benefit analysis it was noted that 95% of nitrate comes from vegetables and approx 5% comes from cured meat. An estimate was made that 135 cases of cancer in the United States result annually from exposure to nitrite, and of these 6 cancers may be attributed to nitrite added to cured meat. The status of the nitrite ban is discussed. Patients should not be concerned that the amount of nitrite they receive in fried bacon or other meat products will cause cancer. (no Refs)

12. COCARCINOGENICITY BY SACCHARIN OF HEPATOCARCINOGENICITY (MEETING ABSTRACT).

Pereira MA
U.S. Enivronmental Protection Agency, Cincinnati, OH
International Symposium on Health Effects of Tumor Promotion, October 12-13, 1981, Cincinnati, Ohio US Environmental Protection Agency and the American College of Toxicology; 1981

We have demonstrated that sodium saccharin was cocarcinogenic towards dibutylnitrosamine (DBN) hepatocarcinogenesis. The simultaneous administration of 5% sodium saccharin in the diet and 0.02% DBN in the drinking water of male rats for 26 wk resulted in a significant increased incidence of preneoplastic foci, hyperplastic nodules and hepatocellular carcinomas compared to rats that received only DBN. The liver of the rats receiving both DBN and saccharin also had an increased incidence of gammaglutamyltranspeptidase (GGTase) positive foci after 4, 8 and 26 wk of treatment. The size distribution of the GGTase-positive foci was the same in the livers of the DBN and DBN-saccharin groups. The livers from rats that only received saccharin did not contain GGTase-positive foci but rather contained at 4 to 8 but not 26 wk a large number of GGTase-positive hepatocytes in Zone 1. It would appear that by 26 wk of saccharin treatment the liver may have developed resistance to saccharin. Our results indicate that the mechanism for the cocarcinogenic activity of saccharin involves an increase in the amount of initiated GGTase-positive foci, and by implication initiation sites of carcinogenesis rather than an increased growth rate of GGTase-positive foci. This implies that the cocarcinogenic activity of saccharin in the liver involves a mechanism different from tumor promoters which act to increase the rate of progression (growth) from the initiated cell to the appearance of cancer. (no Refs)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

13. HEPATOMA AFTER LONG-TERM EXPERIMENTAL ADMINISTRATION OF GRISEOFULVIN.

Chlumska A, Janousek V
U nemocnice 4, 120 000 Praha 2, Czechoslovakia
Cesk Patol; 17(2):83-87 1981

Griseofulvin was administered to 10 white and 2 nude mice for 12 or 14 mo. Diffuse and nodular hepatomas developed. Tumor cells showed atypical ultrastructural changes, particularly in granular endoplasmic reticulum, nucleus, and nucleolus. Results were comparable to those of other carcinogenesis experiments and to human hepatomas. (13 Refs) (In CZE)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

14. EFFECTS OF THE INTERACTION OF DIETARY FAT AND PROTEIN ON EXPERIMENTAL PANCREATIC CANCER IN SYRIAN HAMSTERS (MEETING ABSTRACT).

Birt DF, Pour PM
Eppley Institute for Research in Cancer, University of Nebraska Medical Center, Omaha, NE, 68105
Newsletter Natl Pancreas Cancer Project; VI(2):10 1981

The effects of dietary protein on pancreatic carcinogenesis by N-nitrosobis-(2-oxopropyl)amine (BOP) in the Syrian hamster were evaluated at three dietary fat levels. In previous reports we demonstrated enhanced pancreatic ductular carcinoma yield as the dietary fat level rose. Syrian hamsters were fed three levels of dietary protein (P) as casein [9 low (LP), 18 medium (MP) and 36 high (HP) g/385 Kcal] with each of three levels of fat (F) as corn oil [4.5 low (LF), 9.0 medium (MF) and 18.0 high (HF) g/385 Kcal] either before (3-7 wk of age) or after (8 wk-death) treatment with a single injection (10 mg/kg sc) of BOP. Carcinoma yields were modified by diet only in hamsters fed the various diets after BOP administration. The enhanced carcinoma yield with increasing fat intake after BOP as observed at the MP level was also seen at the HP level in both sexes [HP-LF, 7/47 (15% incidence); HP-HF, 19/55 (35% incidence)]. Effects of dietary protein on carcinoma incidence were observed at the MF level in females [LP-MF, 0/28 (0%); HP-MF, 8/27 (30%)], but not in males [LP-MF, 10/29 (34%); HP-MF, 5/24 (21%)]. In hamsters fed the HF diets the lowest protein level resulted in a decrease in carcinoma yield [LP-HF, 1.5 carcinomas/total body area (TBA); HP-HF, 3.6 carcinomas/TBA]. Pancreatic adenomas were influenced by dietary fat and protein levels before or after BOP treatment. Feeding the low protein diets dramatically reduced adenoma yield, particularly at the high fat level. For example, feeding LP-HF and HP-HF diets to male hamsters resulted in 8 and 14 adenomas/TBA respectively when fed before BOP and 15 and 31 adenomas/TBA when fed after BOP. (no Refs)

15. BIOINTERACTION OF SODIUM SELENITE AND AFLATOXIN B1 IN THE MONGOLIAN GERBIL.

Lalor JH, Llewellyn GC
(c/o Llewellyn) Life Science Building, Virginia Commonwealth Univ., 816-822 Park Ave., Richmond, VA, 23284
J Toxicol Environ Health; 8(3):387-400 1981

Mongolian gerbils were fed diets over a 12-wk period containing one of the following: sodium selenite (SSN: 5.0 ppm), aflatoxin B1 (AFB: 12.8 ppm), or SSN (5.0 ppm) + AFB (12.8 ppm). Controls received commercial Chow. All four groups of animals exhibited significant wt gains, but the gain of those treated with SSN + AFB was significantly less than that in the control group. Both groups treated with AFB drank less water/g body wt than did controls during the first 6 wk. Weekly physical activity analysis showed that the group treated with SSN + AFB was significantly more active than all other groups after 5 wk. Relative to control and SSN groups, the AFB group had lower mean liver, kidney, and lung wt. The SSN + AFB group had decreased liver wt but not kidney or lung wt relative to controls. Histopathological damage was limited primarily to liver and kidney in the three test groups. Gastrointestinal pathology was observed only in the SSN + AFB group. Blood analysis showed no change in total leukocytes, but the relative percentage of lymphocytes increased and of neutrophils decreased concurrently in the order: controls less than AFB less than SSN + AFB less than SSN. Hepatic protein analysis revealed two protein peaks in the SSN + AFB group that were absent in all other groups. The proteins may be selenoproteins which are directly or indirectly involved in the lower incidence of histopathological damage in this group. (41 Refs)

16. THE EFFECT OF DIETARY SELENIUM ON THE METABOLISM AND EXCRETION OF THE HEPATOCARCINOGEN 2-ACETYLAMINOFLUORENE IN THE RAT.

Besbris HJ
University of Southern California, Los Angeles, CA, 90007
Diss Abstr Int [B]; 42(4):1412-B 1981

Hepatic enzymes, presumably involved in the metabolism of the hepatocarcinogen 2-acetylaminofluorene (AAF), were examined in rats fed a Torula yeast-based selenium-deficient diet or the same diet supplemented with 0.5 ppm selenium (as sodium selenite). Selenium-deficient rats exhibited almost total absence of hepatic selenium-dependent glutathione peroxidase activity three wk after initiation of test diet feeding. Dietary selenium had no significant effect on hepatic soluble sulfotransferase activity in vitro as measured with the substrates, N-hydroxy-AAF and phenol. Hepatic lipid peroxidation (HLP) was not altered by AAF-challenge in rats fed either diet. In agreement with previous reports, dietary selenium protected against increased HLP produced by administration of carbon tetrachloride. Selenium-supplemented rats exhibited (a) no significant change in the production of 7-,5-, and N-hydroxy-AAF metabolites and (b) significantly increased production of 3-hydroxy-AAF metabolite as compared to selenium-deficient rats. Dietary selenium had no significant effect on total metabolites excreted or on the major phenolic metabolites excreted into the free-glucosiduronic acid-conjugated, or sulfuric acid-conjugated fractions. However, selenium-deficient rats excreted two and three x more N-hydroxy-AAF into the free-, and glucosiduronic acid-conjugated fractions, respectively. These findings tend to support the hypothesis that dietary selenium alters the in vivo metabolism of AAF in a manner that selectively decreases N-hydroxy-AAF excretion, and possibly its production. Thus dietary selenium's protective effect against AAF-induced hepatocarcinogenesis may be partially explained by the ability of selenium to decrease the amount of N-hydroxy-AAF available for further activation. (no Refs)

17. CHANGE IN HEPATIC GLUTATHIONE (GSH) LEVELS IN MALE AND FEMALE F344 RATS TREATED WITH LOW PROTEIN AND DIETARY AFLATOXIN (AFB1) (MEETING ABSTRACT).

Mainigi KD, Campbell TC
Division of Nutrients, Cornell University, Ithaca, NY
Toxicologist; 1(1):130 1981

It has been proposed that the level of hepatic GSH may play an important role in determining the proportion of procarcinogenic aflatoxin dose that eventually binds to critical cellular mac romolecules. The primary objectives of this study were to investigate the effects of different levels of protein intake and ingestion of hepatotoxic doses of AFB1 on both, hepatic GSH levels and the induction of hepatic lesions. After acclimation to a 20% casein (20C) agar-gel diet for 6 days, groups of weanling male (71g) and female (76g) rats were fed 5C or 20C diets with/without 2.5-5 ppm AFB1 for 6 wk. Animals were sacrificed at selected intervals during this time period and liver GSH concentrations were determined. After the first 12 hr, rats were always killed between 9 and 10 AM to minimize diurnal variations in GSH level. In terms of growth rate, females were quite resistant, both to protein deficiency and dietary AFB1. Males were considerably more responsive to both treatments. On the other hand, the GSH levels in both sexes showed similar response patterns to dietary stresses. All rats on 5C showed decreased levels of GSH, beginning within 3 hr of this treatment. AFB1 increased GSH level regardless of protein status and animal sex. Whereas bile duct proliferation was seen in all AFB1-treated liver, regenerating nodules appeared only in 20C-AFB1 rats. Lesions were more acute in males, but in both sexes they were more pronounced in 20C groups. (no Refs)

18. TUMOR PROMOTION OF THE GASTROINTESTINAL TRACT OF ANIMALS: DIETARY FACTORS (MEETING ABSTRACT).

Newberne PM, Schrager T
Massachusetts Institute of Technology, Cambridge, MA
International Symposium on Health Effects of Tumor Promotion, October 12-15, 1981, Cincinnati, Ohio US Environmental Protection Agency and the American College of Toxicology; 1981

The biological mode of action of tumor promoters exemplified by the phorbol esters, is a subject of intensive study in a number of laboratories. A few investigators have recently begun to examine the role of dietary nutrients in tumor promotion, but the available data are sparse and interpretation difficult. A few examples are provided to indicate that some nutrients may be important in the promotion of cancer. However, the fine dividing line between effects on initiation or on promotion, so clearly shown in the mouse two-stage skin cancer model, is not so clear as yet in models used for studies in nutritional carcinogenesis. The animal models for these studies have been primarily rats, mice and hamsters. These have shown that nutrients which appear to have promotion activity are zinc deficiency and 13-cis-retinoic acid for the esophagus; vitamin A deficiency fat and vitamin A deficiency for liver and colon, lipotrope deficiency for the liver; selenium for the liver. It is probably more correct at this early stage of investigation to consider the effects of nutrients acting either during the time of administration of the carcinogen or, after such administration

and when no detectible carcinogen is found in the animals tissues, rather than as pr omoters in the strict sense. (no Refs)

19. PLASMA ZINC AND VITAMIN A IN HUMAN SQUAMOUS ESOPHAGEAL CANCER (MEETING ABSTRACT).

Mellow MH, Layne EA, Lipman TO, Kaushik M, Hostetler C, Smith JC
Veterans Administration Medical Center, Washington, DC, 20422
Gastroenterology; 80(5,part2):1229 1981

Experimental zinc deficiency in animals promotes esophageal tumor induction with chemical carcinogens. Retinoid administration has suppressed tumor progression in animals. Decreased plasma zinc and vitamin A have been noted in several human squamous cancers. We measured plasma zinc and vitamin A in alcoholic patients with newly diagnosed squamous esophageal cancer and compared these to three age-matched control groups: alcoholics without cancer; non-alcoholics with non-squamous cancer; non-alcoholics without cancer. Patients with factors thought to reduce plasma zinc or vitamin A were excluded: severe liver disease, severe protein energy malnutrition, infection, other acute illness. Conclusions: 1) Alcoholic patients with esophageal cancer have significantly decreased mean plasma zinc values compared to non-alcoholics without cancer. This is unlikely due to alcohol ingestion per se since 6 of 12 alcoholics without cancer had normal zinc values. This is also not due to the presence of a cancer since the non-alcoholics with cancer had normal zinc. 2) Patients with esophageal cancer as well as non-esophageal cancers had low mean plasma vitamin A. Our findings suggest a true deficiency of zinc and/or vitamin A nutrition in patients with esophageal cancer, which may have preceded the onset of illness. Zinc and/or vitamin A deficiency may contribute to the development of esophageal cancer. (no Refs)

20. VITAMIN A STATUS AND LUNG CANCER (MEETING ABSTRACT).

Lopez A, LeGardeur BY, Johnson WD
Department of Medicine, Louisiana State University School of Medicine, New Orleans, LA
Am J Clin Nutr; 34(4):641 1981

Indexes of Vitamin A nutritional status--serum Vitamin A (VITA), serum beta-carotene (CAR), serum retinol-binding protein (RBP), intake of VITA and CAR--were studied in both living patients and autopsied cases with primary lung cancer (LC). Control groups, matched by sex, race, and age, included hospital (H), other-cancer (OC), and free-living (FL) persons. Intake of VITA as Retinol Equivalents (RE) per 1000 KCAL was lower for persons with LC (1332 RE) than controls (1545 RE); however, LC cases vs controls had higher intake of retinol (4.2 mg vs 2.4 mg) and lower of CAR (1.2 mg vs 1.6 mg). In conclusion, results suggest CAR (in addition to VITA) is negatively associated with LC. (no Refs)

21. HIGH DIETARY CELLULOSE: PROTECTION AGAINST LARGE BOWEL CARCINOMA (MEETING ABSTRACT).

Prizont R, Piatt K
Division of Gastroenterology, Albuquerque Veterans Administration Medical Center , Albuquerque, NM
Gastroenterology; 80(5,part2):1256 1981

The effect of high cellulose diet in experimental carcinogenesis, was investigated in Wistar rats injected ip with 10 doses of 1,2 dimethylhydrazine (DMH). Ten experimental animals (ER) were fed a regular diet of casein (20%), sucrose (50%), fat (5%) and cellulose fiber (5%). A second group (EHF) received a similar diet modified by an increase of cellulose (to 15%) and a concomitant 10% decrease of sucrose. Two groups of 6 rats each were injected with buffer and used as animal controls on a regular or high fiber diet. When food consumption was measured, ER animals averaged 20.5 +/-0.3 g/day and EHF rats, 22.7 +/-0.3 g/day. In the same period of time, ER animals had significantly (p less than 0.005) lower fecal outputs than did EHF rats. Rats were sacrificed 7 mo after initial DMH injection. Conversely, 17 intestinal tumors were found in ER animals, which was significantly higher than the 4 recovered from EHF rats. Histological examination revealed that 15 of the 17 ER tumors were malignant (14 adenocarcinomas and 1 signet ring carcinoma). Only 2 of the EHF tumors were adenocarcinomas. The small intestine of ER rats contained 9 tumors (8 adenocarcinomas). The same area in EHF animals yielded 4 tumors (2 malignant). The large intestine of ER rats contained 8 neoplasms (7 malignant), but no benign or malignant tumors were found in the EHF large bowels. This study indicates that DMH injected rats fed a high cellulose diet develop significantly fewer intestine tumors. (1 Ref)

22. FAILURE OF DIETARY CELLULOSE FIBER TO PREVENT COLON TUMORS IN DIMETHYLHYDRAZINE (DMH)-TREATED RATS ON HIGH CHOLESTEROL DIETS (MEETING ABSTRACT).

Tempero MA, West W, Zetterman RK
Department of Internal Medicine, Veterans Administration Medical Center, Omaha, NE, 68105
Gastroenterology; 80(5,part2):1301 1981

A prior study has suggested that dietary fiber (cellulose) may decrease colon carcinogenesis in the DMH-induced rat model. We have evaluated the effect of dietary cellulose fiber on DMH-induced colon tumors in rats fed high cholesterol diets (a suspected co-carcinogen with DMH). Synthetic diets containing excess cholesterol and variable fiber content were fed to male Sprague-Dawley rats: Group I (0% fiber), Group II (5% fiber), and Group III (20% fiber). Half of each group received sc injections of DMH for 20 wk. Control rats received saline. All animals were sacrificed 1 wk after the last injection and blood obtained for cholesterol determination. Rat colons were inspected grossly and microscopically for invasive adenocarcinoma. Saline control animals were tumor free. No difference in either tumor incidence or number of tumors per affected rat colon was observed. Furthermore, the presence of dietary fiber did not alter serum cholesterol level supporting prior observations that cellulose fiber does not affect cholesterol metabolism. These findings suggest excess dietary cholesterol may diminish the protective effect of dietary fiber on DMH-induced colon tumors, possibly through an intraluminal mechanism. (1 Ref)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

23. DIET AND REPRODUCTIVE HORMONES: A STUDY OF VEGETARIAN AND NONVEGETARIAN POSTMENOPAUSAL WOMEN.

Armstrong BK, Brown JB, Clarke HT, Crooke DK, Hahnel R, Masarei JR, Ratajczak T
Unit Epidemiology and Preventive Medicine, Univ. of Western Australia, Nedlands 6009, Western Australia
JNCI; 67(4):761-767 1981

In comparison with matched nonvegetarian women, postmenopausal vegetarian women were found to have lower urinary levels of estriol and total estrogens, lower plasma prolactin levels, and higher plasma sex hormone-binding globulin (SHBG) levels. These differences were not explained by differences in body wt or obesity. Plasma SHBG levels were highly correlated with plasma high-density lipoprotein cholesterol levels, which were also higher in vegetarians than in non-vegetarians. These hormonal differences may explain the lower rates of endometrial and possibly breast cancer that have been observed previously in vegetarian women. (Author abstract) (37 Refs)

24. BILE ACIDS, BUT NOT NEUTRAL STEROLS, ARE TUMOR PROMOTERS IN THE COLON IN MAN AND IN RODENTS (MEETING ABSTRACT).

Weisburger JH, Reddy BS, Wynder EL
American Health Foundation, Valhalla, NY
International Symposium on Health Effects of Tumor Promotion, October 12-15, 1981, Cincinnati, Ohio US Environmental Protection Agency and the American College of Toxicology; 1981

Colon cancer in man has distinct incidence patterns in various parts of the world. The key element distinguishing high and low populations is the intake of dietary fat and fiber. The mechanism whereby dietary fat operates in colon carcinogenesis is through control of endogenous cholesterol biosynthesis which, in turn, is the source of bile acid synthesis in the liver. Bile acids are secreted into the intestinal tract. Primary, and more so secondary bile acids were shown to be colon tumor promoters in several lines of experimentation. Dietary fat, thus, leads to specific total bile acid flow through the intestinal tract, accounting for a promoting effect. Fiber, especially cereal fiber, determines stool bulk which exerts a diluting effect on the total bile acid level in the tract. The concentration of bile acids at the mucosal barrier effectively relates to the extent of promoting action. Those findings have been made in man and animal models, thus strengthening the mechanistic concept underlying colon carcinogenesis. In most areas of the world levels of dietary fat and cholesterol relate to the risk for colon cancer and also for coronary heart disease, the latter through determining the amount of total serum cholesterol and of high density lipoprotein cholesterol. Recent instances of low serum cholesterol and apparent high colon cancer risk are accounted for by our findings, namely a higher conversion of cholestrol to bile acids acting as tumor promoters. Protection in such cases can be readily secured by ensuring an adequate intake of cereal fibers. (no Refs)

25. COMPARISON BETWEEN A PURIFIED AND STANDARD DIET ON FANFT-INDUCED MURINE BLADDER CANCER.

Soloway MS, Nissenkorn I, McCallum LW
Dept. Urology, Univ. Tennessee Center Health Sciences, Memphis, TN
Urology; 18(5):482-484 1981

We investigated the effect of two diets containing equal amounts of the carcinogen FANFT on the incidence and induction time of murine bladder carcinoma in mice. At each time interval, the tumor incidence in the mice ingesting a purified, high-cal diet, was equal or greater than the tumor incidence in the animals fed standard mouse chow. The mean animal wt of the mice ingesting the purified diet was consistently higher than that of the mice fed the standard diet. The bladder wt (an index of tumor vol) showed no significant difference between the mice receiving the two diets. Since mice eat to fulfill their caloric requirements, the animals ate less of the purified diet and the cost per mouse per yr was lower. (Author abstract) (6 Refs)

26. NUTRIENT INTAKES IN RELATION TO CANCER INCIDENCE IN HAWAII.

Kolonel LN, Hankin JH, Lee J, Chu SY, Nomura AM, Hinds MW
Epidemiology Program, Cancer Center Hawaii, Univ. Hawaii, Honolulu, HI, 96813
Br J Cancer; 44(3):332-339 1981

A representative sample of 4,657 adults greater than or equal to 45 yr of age from the five main ethnic groups in Hawaii (Caucasians, Japanese, Chinese, Filipinos and Hawaiians) were interviewed during 1977-1979 regarding their diets. Quantitative food-consumption histories were obtained, from which av daily intakes of fat (saturated, unsaturated, cholesterol, meat, dairy, fish, animal, vegetable and total), protein (animal, meat, fish, dairy and total), carbohydrate, and vitamins A and C (including supplements) were calculated using food-consumption data from standard sources. Multiple regression analysis, with sex as a controlled variable, was used to assess the statistical relationship between these ethnic-sex-specific intakes and corresponding population-based cancer incidence rates of 15 selected sites for which nutrient components are suspected to be either causal or protective. Based on pre-set criteria for establishing important relationships, significant positive associations were found for 6 of the cancer sites: breast cancer with fat (saturated, unsaturated, animal, total) and protein (animal), corpus-uteri cancer with the same components as breast cancer, prostate cancer with fat (saturated, animal) and protein (animal, total), stomach cancer with fat (fish only) and protein (fish only), lung cancer with cholesterol, and laryngeal cancer with cholesterol. Breast and corpus-uteri cancers also showed significant negative associations with carbohydrate intake. The implications of these findings for future research are discussed. (Author abstract) (32 Refs)

27. CANCER OF THE COLON.

Audigier JC, Lambert R
Faculte de medecine, Centre d'Epidemiologie du CNRS LP005440, avenue Rockefeller, 69373 Lyon Cedex 2, France
Gaz Med Fr; 88(28):3903-3909 1981

Epidemiological factors related to colon cancer (CC) are considered. The number of CC cases increases linearly with age for both sexes. The male:female CC ratio is usually greater than 1. The genetic factor is most evident in CC developing in patients with multiple polyposis or extracolonic tumors (Gardner's syndrome). Geographic variations in the incidence

of CC are described. Through the study of persons migrating from Japan or Poland to the United States, a country at high risk for CC, the importance of environment and diet in the development of CC is evident. Variations in CC incidence over time are discussed. The risk of CC is greater in persons with polyps. The malignant potential of these adenomas varies according to the size, histological type, and degree of epithelial abnormality of the lesions. Adenomatous polyps are usually small and less likely to become malignant than are villous polyps. Patients with inflammatory intestinal disease, such as ulcerative colitis and Crohn's disease are more likely to develope CC. An excess of animal fats and cholesterol and a lack of fiber are the dietary factors most often related to the development of CC. The early detection and prevention of CC are discussed. (10 Refs) (In FRE)

28. ASSESSMENT OF THE ROLE OF MUTAGENS AND ENDOGENOUS FACTORS IN LARGE BOWEL CANCER.

Weisburger JH, Spingarn NE, Wang YY, Vuolo LL
American Health Foundation, Valhalla, NY
Cancer Bull; 33(4):124-129 1981

Etiology of large bowel cancer is discussed. The production of abnormal DNA through the direct attack of radiation, chemicals, or viruses, or through improper DNA polymerase activity during DNA synthesis or repair, is only the first step in a long sequence of events terminating in a malignant invasive neoplasm. Rate of cell duplication as controlled by promoters or inhibitors is also important for neoplasm development. Chemical carcinogens, accounting for the majority of human cancers, can be divided into two major groups: genotoxic (direct-acting, procarcinogen, and inorganic carcinogen) and epigenetic (solid-state carcinogen, hormone, immunosuppressor, co-carcinogen, and promoter). Colon cancer incidence and mortality vary as a function of geographic area of residence. The key environmental difference between low-risk and high-risk countries appears related to diet, particulary fat and fiber consumption. In order for colon cancer to occur, both promoters (ie high fat diet) and initiators (ie mutagens produced during frying or broiling of meats and fish) may be required. Variations in these factors and high fiber intake can alter the incidence of colon cancer. (38 Refs)

29. THE RELATIONSHIP BETWEEN DIETARY FAT AND HORMONES IN MAMMARY CARCINOGENESIS (MEETING ABSTRACT).

Dao TL
Roswell Park Memorial Institute, Buffalo, NY
International Symposium on Health Effects of Tumor Promotion, October 12-15, 1981, Cincinnati, Ohio US Environmental Protection Agency and the American College of Toxicology; 1981

Both epidemiologic findings and experimental data provide convincing evidence to support the concept that the etiology of cancer is multifactorial. In experimental mammary carcinogenesis, polypeptide and steroid hormones have been shown to be involved in the initiation and promotion phases of mammary tumor induction. Among the environmental factors suspected to play a role in mammary carcinogenesis, dietary fat correlates well with breast cancer incidence. This paper will present experimental data to demonstrate the co-carcinogenic effect of hormones in the induction of mammary cancer in the rat by polycyclic aromatic hydrocarbons. The experimental data will be compared with human epidemiologic findings. The relationship between dietary fat and mammary carcinogenesis in experimental models will be presented and, finally, the possible relationship between dietary fat and hormones will be discussed. (no Refs)

30. MORTALITY FROM SELECTED MALIGNANT NEOPLASMS IN THE BRITISH ISLES: THE SPATIAL PERSPECTIVE.

Howe GM
Dept. Geography, Univ. Strathclyde, Glasgow, Scotland
Soc Sci Med [Med Geogr]; 15(1):199-211 1981

Standardized mortality ratios (SMRs) for cancer of the trachea, lung, bronchus, stomach, large intestine, and rectum in males and for breast cancer in females (both sexes, age 15-64 yr old) were calculated (1970-72 yr) for 320 administrative units in the United Kingdom. SMRs were also calculated for intestinal and rectal cancer in the Republic of Ireland. The SMRs are shown on demographic maps that demonstrate marked spatial variations in the mortality patterns of the selected neoplasms. Mortality from tracheal, bronchial, and lung cancer was highest in the London borough of Southwark, Glasgow, and Bootle in Lancashire. Gastric cancer mortality was high in North Wales and Scotland, and intestinal cancer mortality was elevated in Dublin, Belfast, and Aberdeen. The spatial distributions did support reported relationships between lung cancer and smoking, between gastric cancer and blood group A, or between intestinal cancer and carbohydrate-rich diets. (27 Refs)

31. FACTORS ASSOCIATED WITH ADENOCARCINOMAS OF THE LARGE BOWEL IN PUERTO RICO.

Martinez I, Torres R, Frias Z, Colon JR, Fernandez N
Cancer Control Program, Dept. Health, Puerto Rico
Rev Latinoam Oncol Clin; 13(1):13-20 1981

A case-control study of 461 patients with adenocarcinoma of the colon seen during the period 1973-1975 and 461 sex- and age-matched controls was conducted. Subjects or relatives (102 pairs) were questioned on long-term dietary and personal habits, and familial aggregation of cancer. Of the 461 adenocarcinomas, 232 were in the colon and 229 were in the rectum and rectosigmoid junction (1.2:1 male-female ratio). Highest number of patients were 65-74 yr old. Twenty-two percent of patients were professionals vs 18.5% of controls. Significantly more patients (29.1%) than controls (16.5%) were heavy cigarette smokers. Patients reported significantly greater frequency of consumption of meats (34.7% high frequency vs 24.3% in controls), cereals (26.7% vs 18.7%), fats (49.2% vs 36.7%), total residue (36.4% vs 29.5%), and fiber (51.4% vs 42.3%). Patients also had more previous nonmalignant chronic disease of the large bowel (diverticulosis, intestinal polyposis, ulcerative colitis, other types of colitis, chronic constipation, diarrhea, and hemorrhoids), higher grade of constipation, and more direct relatives with cancer (for cancer of the large bowel, 22 relatives among patients vs 9 among controls). Recall bias may be expected in this type of study. (25 Refs)

32. ADVANCES IN CARCINOMA OF THE OESOPHAGUS.

Silber W
Dept. Surgery, Medical Sch., Observatory, Cape, South Africa
S Afr Cancer Bull; 25(1/2):24-30 1981

Epidemiology, etiology, diagnosis, and treatment of esophageal carcinoma (EC) are discussed. Incidence of EC can vary 500-fold from one geographic area to another. The highest incidence of EC occurs in economically less advanced countries where population data are inadequate and cancer registration is difficult. In view of the increased incidence of EC in the Cape Colored male, the life style of this group is being studied with emphasis on drinking and smoking habits and other hazards to

which members may be exposed. EC seems to be multifactorial in origin. Risk factors, such as tobacco and alcohol, are considered. Factors related to increased incidence of EC in Transkei, including level of minerals in rocks, soil, or plants, low levels of plant nutrients, manganese deficiency-induced vitamin C deficiency in plants, fungus infected maize, and nitrosamine levels are considered. A gross deficiency of serum beta-carotene has been found in an asymptomatic Black male group from a high-risk area in Transkei. Besides prevention, early diagnosis is the only method for improving the otherwise poor diagnosis for EC. Screening and dia gnostic techniques for EC are described. Therapeutic techniques for EC are discussed. (14 Refs)

33. TUMOURS IN ICELAND: 4. TUMOURS OF THE UPPER RESPIRATORY TRACT AND EAR. A HISTOLOGICAL CLASSIFICATION AND SOME ETIOLOGICAL AND EPIDEMIOLOGICAL CONSIDERATIONS.

Cooper MA, Hallgrimsson J
Dept. Pathology, Univ. Iceland, 121 Reykjavik, Iceland
Acta Pathol Microbiol Scand [A]; 89(5):377-387 1981

Primary tumors from the upper respiratory tract and ear submitted for a histological diagnosis in Iceland during the 20 yr, 1955-1974, were reviewed and typed according to the World Health Organization Classification published in 1978. The series included 83 benign and 114 malignant tumors from the upper respiratory tract. The nasal cavity was the commonest site for benign tumors in both sexes, most being squamous cell and transitional papillomas. The larynx was the commonest site for malignant tumors in males, all being squamous cell carcinomas, and the paranasal sinuses and the nasopharynx in females, squamous cell carcinoma forming the largest group in the sinuses and undifferentiated carcinoma in the nasopharynx. The distribution of tumors in the nasal cavity and sinuses differed much between the two sites and between the sexes, which emphasizes that these two sites should be separated and not combined as they have been until recently in epidemiological reports and cancer registries. A relatively high incidence of nasopharyngeal carcinoma in Iceland may be due to a high consumption of salted food. A rising incidence of laryngeal carcinoma is believed to be related to the use of tobacco and alcohol and is of special concern owing to its marked rise in young females. (Author abstract) (30 Refs)

GENERAL REVIEWS AND OTHER STUDIES

34. NUTRITION AND ENVIRONMENTAL HEALTH: THE INFLUENCE OF NUTRITIONAL STATUS ON POLLUTANT TOXICITY AND CARCINOGENICITY. VOLUME 2: MINERALS AND MACRONUTRIENTS.

Calabrese EJ
Environmental Health Program, Div. Public Health, Univ. Massachusetts, Amherst, MA
Nutrition and Environmental Health: The Influence of Nutritional Status on Pollutant Toxicity and Carcinogenicity. Volume 2: Minerals and Macronutrients. New York, John Wiley and Sons, 468 pp., 1981.

As part of a survey on the influence of nutritional status on pollutant toxicity and/or carcinogenicity, the roles of minerals (calcium, copper, iron, selenium, zinc, cobalt, iodine, magnesium, manganese, molybdenum, phosphorus, potassium, and sulfur), protein, fats, carbohydrates, specific amino acids, fiber, and synthetic antioxidants are detailed. A comprehensive, critical review of the literature and viable and socially relevant research hypotheses to be considered in the formulation of priority allocations are presented. Persons who may be at increased risk for development of adverse effects from pol lutant exposures are indicated. (2115 Refs)

35. THE NATIONAL LARGE BOWEL CANCER PROJECT: OVERVIEW.

Copeland MM
M. D. Anderson Hospital and Tumor Inst., Univ. Texas, Houston, TX
Cancer Bull; 33(4):121-123 1981

Areas of research supported by the National Large Bowel Cancer Project are discussed. The incidence, diagnosis, and treatment of colon cancer are considered. Topics discussed in relation to large bowel cancer include the availability of suitable animal models, causation and biochemical control factors in cellular metabolism, relationship between diet and large bowel cancer, isolation and identification of mutagens/carcinogens resulting from cooking of meats and other products, chemotherapy and immunotherapy, colon cancer screening, and familial polyposis and Gardner's syndrome as models for colon carcinoma. (31 Refs)

36. ON THE CAUSES AND PREVENTION OF GASTROINTESTINAL CANCERS (MEETING ABSTRACT).

Weisburger JH
American Health Foundation, Valhalla, NY, 10595
Gastroenterology; 80(5,part2):1313 1981

The decreased incidence of gastric cancer in the United States can be traced to a lower level of gastric carcinogen ingestion as a result of lesser use of pickling and greater availability on a year-round basis of foods containing vitamin C. Colon cancer is the neoplastic disease with the highest incidence in men and women in the United States. The incidence has increased only slightly in the last 50 yr. Current evidence indicates that the disease has several risk factors. The genotoxic carcinogen may be in the brown part of fried food which is shown to be highly mutagenic and in turn contains several components. Addition of soy protein inhibits the information of mutagenic materials and may be one approach to prevention. The level of total dietary fat controls the amount of biosynthetic bile acids in the gut which, in turn, have promoting activity. The concentration of bile acids depends on dietary fat ingested and stool bulk, in turn a function of dietary fiber from cereal grains. Increasing fat raises the risk, whereas increasing fiber lowers the risk. Thus, parallel concordant results from studies in man and in animal models provide a suggestive data base for approaches to the prevention of colon cancer through 1) studying the inhibition of the formation of genotoxic carcinogens in the brown part of fried food, 2) lowering the total amount of dietary fat intake, and 3) increasing the amount of fiber intake. (no Refs)

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

DIETARY FACTORS IN CARCINOGENESIS

1. NUTRITION AND ENVIRONMENTAL HEALTH: THE INFLUENCE OF NUTRITIONAL STATUS ON POLLUTANT TOXICITY AND CARCINOGENICITY. VOLUME 2: MINERALS AND MACRONUTRIENTS.

Calabrese EJ

Environmental Health Program, Div. Public Health, Univ. Massachusetts, Amherst, MA

Nutrition and Environmental Health: The Influence of Nutritional Status on Pollutant Toxicity and Carcinogenicity. Volume 2: Minerals and Macronutrients. New York, John Wiley and Sons, 468 pp., 1981.

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

2. RETROSPECT AND PROSPECT OF STUDIES ON DIGESTIVE SYSTEM DISEASES IN CHINA.

Zhang X

Dept. Internal Medicine, Capital Hosp., Chinese Acad. Medical Sciences, Beijing, People's Republic of China

Zhonghua Xiaohua Zazhi; 1(1):1-4 1981

In CHI

3. EPIDEMIOLOGY OF PITUITARY ADENOMAS.

Gold EB

Dept. Epidemiology, Sch. Hygiene and Public Health, Johns Hopkins Univ., 615 Wolfe St., Baltimore, MD, 21205

Epidemiol Rev; 3:163-183 1981

4. BLADDER CANCER EPIDEMIOLOGY.

Mananoski GM, Elliott EA

Dept. Epidemiology, Sch. Hygiene and Public Health, Johns Hopkins Univ., 615 North Wolfe St., Baltimore, MD, 21205

Epidemiol Rev; 3:203-229 1981

5. THE EPIDEMIOLOGY OF BENIGN BREAST DISEASE.

Ernster VL

Dept. Epidemiology and International Health, 1699HSW, Univ. California, San Francisco, CA, 94143

Epidemiol Rev; 3:184-202 1981

ENVIRONMENTAL AND OCCUPATIONAL FACTORS IN CARCINOGENESIS

6. AIR POLLUTION AND BIOLOGICAL DEFENCE.

Schlipkoter HW, Dolgner R

Med. Institut fur Umwelthygiene an der Univ., Gurlittstrasse 53, D-4000 Dusseldorf 1, W. Germany

Zentralbl Bakteriol [Orig A]; 172(4/5):299-311 1981

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7. CANCER AND THE PHYSICOCHEMICAL QUALITY OF DRINKING WATER IN QUEBEC.

Thouez JP, Beauchamp Y, Simard A

Dept. Geography, Univ. Montreal, Montreal, Quebec, Canada

Soc Sci Med [Med Geogr]; 15(1):213-223 1981

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

8. ONCOGENIC VIRUSES.

Wyke JA

Imperial Cancer Res. Fund, P.O. Box 123, Lincoln's Inn Fields, London WC2A 3PX, England

J Pathol; 135(1):39-85 1981

9. ROLE OF VIRUSES IN THE ETIOLOGY OF NATURALLY OCCURRING FELINE LEUKEMIA.

Essex M, Grant CK, Cotter SM, Hardy Jr WD

Dept. Microbiology, Harvard Sch. Public Health, 665 Huntington Ave., Boston, MA, 02115

Haematol Blood Transfus; 26:483-487 1981

METHODOLOGY IN CARCINOGENESIS RESEARCH

10. MAMMALIAN IN VIVO AND IN VITRO CYTOGENETIC ASAYS: A REPORT OF THE U.S. EPA'S GENE-TOX PROGRAM.

Preston RJ, Au W, Bender MA, Brewen JG, Carrano AV, Heddle JA, McFee AF, Wolff S, Wassom JS

Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830

Mutat Res; 87(2):143-188 1981

11. BACTERIAL SYSTEMS FOR CARCINOGENICITY TESTING.

Mohn GR

Dept. Radiation Genetics and Chemical Mutagenesis, State Univ. Leiden, Wassenaarseweg 72, 2333 AL Leiden, The Netherlands

Mutat Res; 87(2):191-210 1981

12. MUTAGENESIS BY CHEMICAL AGENTS IN V79 CHINESE HAMSTER CELLS: A REVIEW AND ANALYSIS OF THE LITERATURE.

Bradley MO, Bhuyan B, Francis MC, Langenbach R, Peterson A, Huberman E

Merck Inst. for Therapeutic Res., West Point, PA, 19486

Mutat Res; 87(2):81-142 1981

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13. CANCER BIOLOGY.

Ruddon RW

Biological Markers Program, NCI-Frederick Cancer Res. Center, Frederick, MD

Cancer Biology. New York, Oxford University Press, 344 pp., 1981.

14. AVOIDABLE CANCER: ATTRIBUTION OF RISK.

Doll R

Radcliffe Infirmary, Oxford, England

S Afr Cancer Bull; 25(3):125-146 1981

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DIETARY ASPECTS OF CARCINOGENESIS

SERIES CK03 No. 82/04

APRIL, 1982

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- GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS..... 7-10
- GENERAL REVIEWS AND OTHER STUDIES..... 11-14

STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. A STUDY ON CARCINOGENESIS INDUCED BY DEGRADED CARRAGEENAN ARISING FROM SQUAMOUS METAPLASIA OF THE RAT COLORECTUM.

Oohashi Y, Ishioka T, Wakabayashi K, Kuwabara N
First Dept. Pathology, Juntendo Univ. Sch. Medicine, 2-1-1 Hongo, Bunkyo-ku, Tokyo, Japan
Cancer Lett; 14(3):267-272 1981

Carcinogenesis in the colorectum of F344 rats fed diets containing 10% degraded carageenan for varying lengths of time was investigated. After termination of carageenan administration, all rats were fed a basal diet until sacrifice 18 mo after initial administration. In males treated for 2 mo, the incidence of colorectal squamous metaplasia was 100%, with tumors in

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5/39 animals. The tumors were squamous cell carcinoma, anaplastic carcinoma, and adenoma. In males treated for 6 mo, metapla sia was evident in 100% and 8/42 rats had tumors. In males treated for 9 mo, 100% of animals showed squamous metaplasia and 17/42 had tumors. No inflammatory or neoplastic changes were found in control animals. (10 Refs)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

2. COFFEE AND OVARIAN CANCER.

Trichopoulos D, Papapostolou M, Polychronopoulou A
Dept. Hygiene and Epidemiology, Univ. Athens Medical Sch., Goudi, Athens 609, Greece
Int J Cancer; 28(6):691-693 1981

Ninety-two women with common epithelial tumors of the ovary and 105 comparison women admitted for various orthopedic conditions were interviewed regarding demographic, socioeconomic and biomedical characteristics, including their use of coffee and tobacco. There was no association between tobacco smoking and ovarian cancer. However a statistically significant trend association was noted between coffee drinking and ovarian cancer (p = approx 0.03). The association could not be explained in terms of any other variable included in the present study. The relative risk for women drinking two or more cups of coffee per day was 2.2 with 95% confidence limits 1.0-4.8. Several descriptive epidemiologic data are compatible with a causal association between coffee and ovarian cancer. (Author abstract) (18 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

3. DIETARY VITAMIN A AND RISK OF CANCER IN THE WESTERN ELECTRIC STUDY.

Shekelle RB, Liu S, Raynor WJ, Lepper M, Maliza C, Rossof AH, Paul O, MacMillan Shryock A, Stamler J
Dept. Preventive Medicine, Rush-Presbyterian-St Luke's Medical Center, Chicago, IL, 60612
Lancet; 2(8257):1185-1190 1981

Intake of dietary provitamin A (carotene) was inversely related to the 19-yr incidence of lung cancer in a prospective epidemiological study of 1,954 middle-aged men. The relative risks of lung cancer in the first (lowest) to fourth quartiles of the distribution of carotene intake were respectively, 7.0, 5.5, 3.0, and 1.0 for all men in the study, and 8.1, 5.6, 3.9, and 1.0 for men who had smoked cigarettes for 30 or more yr. Intake of preformed Vitamin A (retinol) and intake of other nutrients were not significantly related to the risk of lung cancer. Neither carotene nor retinol intake was significantly related to the risk of other carcinomas grouped together, although for men in whom epidermoid carcinomas of the head and neck subsequently developed, carotene intake tended to be below av. These results support the hypothesis that dietary beta-carotene decreases the risk of lung cancer. However, cigarette smoking also increases the risk of serious diseases other than lung cancer, and there is no evidence that dietary carotenoids affect these other risks in any way. (Author abstract) (23 Refs)

4. CHEMOPREVENTION OF CANCER WITH RETINOIDS.

Peck GL
Dermatology Branch, National Cancer Inst., Bulding 10, Room 12N238, Bethseda, MD, 20205
Gynecol Oncol; 12(2,part2):S331-S340 1981

Use of synthetic retinoids, isotretinoin (13-cis-retinoic acid, RO-4-378, Accutane) and an aromatic retinoid (etretinate, RO-10-9359, Tigason), in cancer prevention and therapy for cutaneous and internal tumors was reviewed. Etretinate and oral isotretinoin were used in treatment of basal cell carcinoma Etretinate was used in treatment of lesions of epidermodysplasia verruciformis induced by the oncogenic human papilloma virus (types 3 and 5), flat, genital, and plantar warts. Cutaneous metastases of malignant melanoma are tested with retinoids. Leukoplakia of the oral cavity, hyperkeratosis of the larynx, and papillomas of the urinary bladder have been treated with etretinate. Chemoprevention of cancer has been accomplished experimentally with chemically diverse agents (carotenoids, folic acid, butylated hydroxytoluene, butylated hydroxyanisole, selenium, ethoxyquin, disulfiram, vitamin E) Retinoids are known to inhibit tumor promotion. Predominant observed toxicities of synthetic retinoids in man involve the skin and mucous membranes. Systemic toxicities are arthralgia, temporary low-grade elevation of serum transaminases and teratogenicity. Observed acute toxicities were well tolerated and no chronic toxicities have been observed. (55 Refs)

5. INCREASE IN FAECAL NITROSAMINES IN JAPANESE INDIVIDUALS GIVEN A WESTERN DIET.

Suzuki K, Mitsuoka T
Animal Physiology Lab., Inst. Physical and Chemical Res., Wako-shi, Saitama 351, Japan
Nature; 294(5840):453-456 1981

Levels of five volatile nitrosamines were studied in six healthy Japanese men (27-39 yr old) given Japanese and Western diets. Three subjects received, in order: (1) a typical Japanese diet for 4 days; (2) a mixed Japanese diet (ie, items from Western, Chinese, and typical Japanese diets) for 3 days; (3) a balanced Western-style diet for 8 days followed by a typical Japanese diet for 6 days. Four subjects received a long-term mixed Japanese diet followed by a high-fat diet containing a large proportion of meat for 4 days. The av level of total volatile nitrosamines in the feces of subjects receiving the Western-style diet was 10x that in feces of subjects receiving the Japanese diet. The max level of total nitrosamines was 26.6 ug/kg. Feces of subjects receiving the Japanese diet contained N-nitroso-dimethylamine (NDMA), N-nitrosodibutylamine (NDBA), and N-nitrosopyrrolidine (NPYR) at levels less than 1.7 ug/kg. After 3 days on the mixed Japanese diet, N-nitrosopiperidine (NPIP) appeared in the feces, and the NPYR concentration increased. When the subjects were given the Western diet, the levels of these nitrosamines increased markedly, particularly NPIP, which reached a max of 13.3 ug/kg. The max level of fecal N-nitrosodiethylamine was 1.8 ug/kg during the experiment, and there was no clear relationship between the level of this nitrosamine and change in diet. Nitrosamine levels decreased when subjects were receiving the high-fat, high-meat diet. These results suggest that fecal nitrosamines are formed in the intestine from precursors contained in meat and vegetables. (27 Refs)

6. DIETARY VITAMIN C AND UTERINE CERVICAL DYSPLASIA.

Wassertheil-Smoller S, Romney SL, Wylie-Rosett J, Slagle S, Miller G, Lucido D, Duttagupta C, Palan PR
Dept. Community Health, Albert Einsten Coll. Medicine, Rm. 1308A, 1300 Morris Park Ave., Bronx, NY, 10461
Am J Epidemiol; 114(5):714-724 1981

A case-control study of women with cervical abnormalities identified through Pap smears, was conducted in the Bronx, New York, to explore the relationship between nutritional intake and cervical dysplasia. Nutrient intake was estimated from computer analysis of 3-day food records and 24-hr recall for 169 study participants (87 cases, 82 controls), including a subset of 49 pairs matched for age, race and parity. Mean vitamin C intake per day from 3-day food record for controls was 107 mg, compared to 80 mg for cases (p less than 0.01). Analysis of matched pairs showed similar results; 29% of cases compared to 3% of controls in matched subset had vitamin C intake less than 50% of the recommended daily allowance, yielding a 10-fold increase in risk of cervical dysplasia as estimated by odds ratio (p less than 0.05). Younger age, greater frequency of sexual intercourse and younger age at first intercourse were associated with higher risk of cervical dysplasia. Multiple logistic analyses indicated that low vitamin C intake is an independent contributor to risk of severe cervical dysplasia when age and sexual activity variables are controlled. Approx 35% of US women in their reproductive yr have daily vitamin C intake below 30 mg, and 68% have vitamin C intake below 88 mg. If other studies confirm these findings, it may be important to explore a possible protective role of supplementary vitamin C for women at high risk of cervical cancer. (Author abstract) (31 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

7. CHANGES IN JAPANESE EATING HABITS AND CANCER OF THE LARGE INTESTINE.

Narisawa T
First Dept. Surgery, Akita Univ. Sch. Medicine, Akita, Japan
Nippon Rinsho; 39(5):26-31 1981

Relationship between increasing rates of cancer of the large intestine and changes in eating habits by Japanese was studied with the aid of experimental animal models and review of literature. Epidemiological studies indicate that environmental and lifestyle differences account for increasing incidence of large intestine cancer more than genetic or anthropological factors. Data from experiments on producing cancer of the large intestine in rats fed either high protein (40%) and high fat (20%) die ts or standard diets (20% protein, 5% fat) along with carcinogenic substances such as 1,2-dimethylhydrazine or azoxymethan supported epidemiological studies indicating that high fat and protein diets were related to high rates of cancer. Other studies on relationship of fiber content in the diet and cancer of the large intestine suggested that prolonged retention of fecal matter in the intestinal lumen increased the concentration and carcinogenic potential of secondary bile acids in fecal matter. Japanese dietary intake of fats has increased 2.2x in the past 20 yr, and concurrent with a rise in consumption of meat (50% of protein intake), the fats consumed are increasingly from animal sources instead of vegetable sources. With decreased consumption of grains, the fiber intake of Japanese had also diminished, reducing the amount of fecal matter and increasing the concentration of bile acids. (32 Refs) (In JPN)

8. EPIDEMIOLOGICAL VICISSITUDES OF LARGE INTESTINE CANCER AND OUTLOOK FOR THE FUTURE.

Hirayama T
Dept. Epidemiology, Natl. Cancer Res. Center, Tokyo, Japan
Nippon Rinsho; 39(5):10-20 1981

Age-adjusted death rates for transverse colon cancer and rectal cancer increased from 1950-1978 in Japan. In 1950 and 1978, respectively, death rates (per 100,000 population) for transverse colon cancer were 1.7 and 3.8 for men and were 2.2 and 4.1 for women; those for rectal cancer were 2.8 and 3.8 for men and 2.5 and 3.0 for women, respectively. Analysis of annual transitions of death rates by age groupings showed that death rates due to transverse colon cancer increased from 1950-1978 in all age groups, and the trend was slightly higher for men than women; except for women 40-44 yr old, death rates due to rectal cancer remained the same or decreased slightly for both men and women. By region, both age-adjusted death rates and rates of chronic disease due to transverse colon cancer showed marked increases for both men and women, and rates of increase of chronic disease due to transverse colon cancer are now 2x those for lung cancer in both men and women. Autopsy studies from 1968-1978 showed that the incidence of cancers in the sigmoid flexure increased 3.1x in men and 2.6x in women. Cigarette smokers had high death rates due to large intestine cancer, and meat eaters had low rates of death due to large intestine cancer. Extrapolation of current data indicates that death rates due to large intestine cancer will continue to rise in the future. Preventative effects of fibrous foods need to be investigated as one aspect of research on changing this trend. (no Refs) (In JPN)

9. DIETARY FACTORS AND BREAST CANCER RISK.

Lubin JH, Burns PE, Blot WJ, Ziegler RG, Lees AW, Fraumeni JF
Environmental Epidemiology Branch, NCI, Bethesda, MD
Int J Cancer; 28(6):685-689 1981

As part of a case-control study in northern Alberta, Canada, 577 women aged 30-8 0 with breast cancer diagnosed during 1976-77 and a population-based age-stratified random sample of 826 disease-free female controls were questioned about certain aspects of their diet. Computing relative risks (RRs) by tertiles, significant increasing trends were found with more frequent consumption of beef (RRs of 1.0, 2.3, 1.5; test for trend, p less than 0.001), pork (RRs of 1.0, 1.6, 2.2; test for trend, p less than 0.001), and sweet desserts (RRs of 1.0, 1.3, 1.5; test for trend, p = 0.01). Elevated risks were also noted for use of butter at the table and for frying with butter or margarine, as opposed to vegetable oils. The association of total beef and pork consumption with breast cancer was not materially affected by controlling for age at first birth, family history of breast cancer, previous benign breast biopsy or socioeconomic status. Nor was the association reduced by controlling for ages of menarche and menopause, even though within the control series the intake of beef and pork reported in adult life was higher among those with a lower age at menarche or an older age at natural menopause. (Author abstract) (26 Refs)

10. ROLE OF NUTRITIONAL FACTORS IN REALIZATION OF BIOLOGICAL EFFECTS OF XENOBIOTICS.

Sharmanov TS
Kazakh Branch, Inst. Nutrition, Alma-Ata, USSR
Vopr Pitan; (5):51-57 1981

The role of nutritional factors in the metabolism of pesticides, nitrosamines, and aflatoxins is reviewed. Nitrites present in canned meat or fish can react with exogenous amines to yield carcinogenic nitroso compounds. Dietary deficiency of vitamin C and essential amino acids enhanced the carcinogenic activity of N-nitrosopyperidine in guinea pigs. Deficiency of the essential amino acids and retinol enhanced the incidence of aflatoxin B1-induced tumors in rats. (33 Refs) (In RUS)

GENERAL REVIEWS AND OTHER STUDIES

11. CAFFEINE INGESTION DURING PREGNANCY: IN UTERO EXPOSURE AND POSSIBLE EFFECTS.

Soyka LF
Clinical Pharmacology, Mead Johnson and Co., 2404 Pennsylvania Ave., Evansville, IN, 47721
Semin Perinatol; 5(4):305-309 1981

Mutagenic significance of caffeine in man and results of animal studies are reviewed. Topics include sources of caffeine exposure, absorption and distribution, mutagenicity, carcinogenicity, teratogenicity, influence on labor and delivery, aminophylline and lung maturation, and fetal breathing. Data on the caffeine content of beverages as consumed in the home, caffeine levels (term amniotic fluid, term cord blood, serum during therapy), and elimination of caffeine from serum of different population groups are summarized. Possible long-term hazards of caffeine exposure are listed. No convincing evidence exists for carcinogenicity of caffeine in animals. Teratologic effects of caffeine in more than 30 studies in animals were only found with high doses (250-800 mg/kg) toxic to the mother. In 10 studies using less than 50 mg/kg caffeine, congenital malformations were not increased. Both caffeine and theophylline have markedly prolonged and variable half-lives, especially in the premature infant; prolonged effects from these drugs cannot be dismissed. (29 Refs)

12. CHEMICAL AND ENVIRONMENTAL AGENTS IN GASTROINTESTINAL CARCINOGENESIS.

Deschner EE, De Cosse JJ, Sherlock P
Lab. Digestive Tract Carcinogenesis, Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY, 10021
Clin Gastroenterol; 10(3):755-771 1981

A review is presented of incidence, risk factors, and chemical and environmental agents involved in induction or promotion of cancer of the gastrointestinal system. Presence of ingested agents in foods and experimental and clinical evidence for cancer causation are discussed. Carcinogenesis is considered with respect to the esophagus (nitrosamines, mycotoxins, promotional factors), stomach (nitrites and polycyclic hydrocarbons, mycotoxins, agricultural products, polychlorinated biphenyls, experimental carcinogens), and large bowel (nitrites, aflatoxins, diet, carrageenan, chemical carcinogens: nitroso compounds, biphenyls, symmetrical 1,2-dialkylhydrazines and azoalkanes and azoxyalkanes). Chemicals with known gastric tumor induction capability in animals are tabulated from published reports. (74 Refs)

13. NUTRITION AND CANCER: A REVIEW.

Muller E, Rickenbach M
Institut fur Sozial- und Praventivmedizin, Universitat Zurich, Gloriastrasse 32 B, 8006 Zurich, Switzerland
Praxis; 70(43):1903-1912 1981

The possible roles of various nutrients in carcinogenesis are reviewed. Epidemiological studies indicate a high rate of carcinomas of the breast (BC) and colon (CC) in industrialized countries. Dietary factors are suggested by changes in cancer frequencies among immigrant populations and their descendants. Dietary studies indicate a positive correlation between dietary fat and CC and BC, as well as a negative correlation between these cancers and dietary fiber content. Animal studies have shown a cancer-promoting effect for fats, particularly unsaturated fats. Fats may promote cancer by affecting specific intestinal bacterial populations, bile secretion, hormonal balance, and immune response. Fiber may reduce cancer risk by speeding the movement of carcinogens through the digestive tract. Cancer risk has not been confirmed for proteins or their pyrolysis products. Some studies have indicated risk associated with pickled, smoked, or fried foods related to production of nitrosamines and nitrosamides. These compounds, which have been shown to be carcinogenic, are formed in vitro from nitrites and secondary amines. Their formation appears to be limited by the presence of ascorbic acid or vitamin E. Further studies of dietary factor s are indicated. (130 Refs) (In GER)

14. RETHINKING THE ENVIRONMENTAL CAUSATION OF HUMAN CANCER.

Higginson J
International Agency Res. on Cancer, 150 cours Albert Thomas, 69372 Lyon Cedex 2, France
Food Cosmet Toxicol; 19(5):539-548 1981

Epidemiologic studies concerning the etiology of environmental cancers are reviewed. The results of a number of studies strongly suggest that most human cancers are related to environmental factors and not to racial or hereditary factors. The results of the author's survey in South Africa further suggested that 'way of life' was of major significance for many cancers, especially those of the gastrointestinal tract and endocrine-dependent organs. Although there are many factors involved in its etiology, cancer may be prevented by controlling a single predominant factor. Only a small part of the total cancer burden can be directly related to ambient environmental pollution, and a number of other environmental factors, particularly diet, must be considered for the majority of environmental cancers. Although the role of dietary factors in promotion and enzyme induction of cancers is well established in laboratory animals, their role in man is much less clear. Most epidemiologists do not consider human environmental cancers in terms of single causes; human cancers for which there is evidence of multistage and multifactorial origin include lung, esophagus, liver, stomach and large intestine, breast and endometrium, Burkitt's lymphoma, and nasopharyngeal carcinoma. (106 Refs)

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As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. CHEMICAL CARCINOGENESIS.
Farber E
Dept. Pathology, Univ. Toronto, 100 College St., Toronto, ON M5G 1L5, Canada
N Engl J Med; 305(23):1379-1389 1981

2. N-HEXANE AND ITS TOXICOLOGIC EFFECTS. A REVIEW.
Jorgensen NK, Cohr KH
Harevaenget 44, DK-4200 Slagelse, Denmark
Scand J Work Environ Health; 7(3):157-168 1981

3. SAFROLE: ITS METABOLISM CARCINOGENICITY AND INTERACTIONS WITH CYTOCHROME P-450.
Ioannides C, Delaforge M, Parke DV
Dept. Biochemistry, Univ. Surrey, Guildford, Surrey GU2 5XH, England
Food Cosmet Toxicol; 19(5):657-666 1981

RADIATION CARCINOGENESIS

4. CARCINOGENIC, MUTAGENIC, TERATOGENIC WORKSHOP.
Suit H, Hall E, Frei E
Dept. Radiation Medicine, Massachusetts General Hosp., Boston, MA, 02114
Cancer Clin Trials; 4(Suppl):37-41 1981

5. DOSIMETRY OF THE ATOMIC BOMB SURVIVORS.
Sinclair WK, Failla P, Bond VP, Loewe WE, Kerr GD, Auxier J, Wyckoff OH, Maruyama T, Schull JW, Land EC, Dobson LR, Mays WC, Seymour J
Argonne Natl. Lab., 9700 South Cass Ave., Argonne, IL, 60439
Radiat Res; 88(3):437-447 1981

DIETARY FACTORS IN CARCINOGENESIS

6. NUTRITION AND CANCER: A REVIEW.
Muller E, Rickenbach M
Institut fur Sozial- und Praventivmedizin, Universitat Zurich, Gloriastrasse 32 B, 8006 Zurich, Switzerland
Praxis; 70(43):1903-1912 1981
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7. FLAVONOIDS, MUTAGENS, AND CITRUS.
Horowitz RM
U. S. Dept. Agriculture, Agricultural Res. Service, Fruit and Vegetable Chemistry Lab., Pasadena, CA, 91106
ACS Symp Ser; 170:43-59 1981

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

8. ETIOLOGICAL CONCEPTS IN CERVICAL CARCINOGENESIS.
Kessler II
Dept. Epidemiology and Preventive Medicine, Univ. Maryland Sch. Medicine, 655 West Baltimore St., Baltimore, MD, 21201
Gynecol Oncol; 12(2,part2):S7-S24 1981

9. CURRENT CONCEPTS ON THE AETIOPATHOGENESIS OF ACUTE LEUKAEMIA.
Torres A, Martinez Guibelalde F, Gomez Garcia P, Garcia Castellano JM, Manzanares Escribano MR
Jefe Servicio Hematologia, Ciudad Sanitaria 'Reina Sofia' de la Seguridad Social, Cordoba, Spain
Sangre (Barc); 26(5B):700-717 1981
In SPA

10. CANCER FAMILY SYNDROME.
Svendsen LB, Bulow S
Kirurgisk-gastroenterologisk afdeling, Bispebjerg hospital, Copenhagen, Denmark
Ugeskr Laeger; 143(42):2713-2716 1981
In DAN

11. GENETIC AND BIOCHEMICAL STUDIES WITH ATAXIA TELANGIECTASIA.
Huang PC, Sheridan RB
Dept. Biochemistry, Sch. Hygiene and Public Health, Johns Hopkins Univ., Baltimo re, MD, 21205
Hum Genet; 59(1):1-9 1981

ENVIRONMENTAL AND OCCUPATIONAL FACTORS IN CARCINOGENESIS

12. THE HEALTH EFFECTS AND ECOLOGICAL SIGNIFICANCE OF CHLORINE RESIDUALS IN WATER.
Kalmaz EV, Kalmaz GD
Univ. Tennessee, Univ. Station 8163, Knoxville, TN, 37916
Chemosphere; 10(10):1163-1175 1981

13. CHEMICAL AND ENVIRONMENTAL AGENTS IN GASTROINTESTINAL CARCINOGENESIS.
Deschner EE, De Cosse JJ, Sherlock P
Lab. Digestive Tract Carcinogenesis, Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY, 10021
Clin Gastroenterol; 10(3):755-771 1981

CELLULAR AND MOLECULAR MECHANISMS IN CARCINOGENESIS

14. THE REPAIR OF DNA DAMAGE: RECENT DEVELOPMENTS AND NEW INSIGHTS.
Friedberg EC, Bonura T, Love JD, McMillan S, Radany EH, Schultz RA
Dept. Pathology, Lab. Experimental Oncology, Stanford Univ., Stanford, CA, 94305
J Supramol Struct Cell Biochem; 16(1):91-103 1981

MODIFICATION OF CARCINOGENESIS

15. ROLE OF HORMONES INCLUDING DIETHYLSTILBESTROL (DES) IN THE PATHOGENESIS OF CERVICAL AND VAGINAL INTRAEPITHELIAL NEOPLASIA.
Robboy SJ, Truslow GY, Anton J, Richart RM
Dept. Pathology, Massachusetts General Hosp., Boston, MA, 02114
Gynecol Oncol; 12(2,part2):S98-S110 1981

16. PROSTAGLANDINS AND CANCER: A REVIEW OF TUMOR INITIATION THROUGH TUMOR METASTASIS.
Honn KV, Bockman RS, Marnett LJ
Dept. Biological Sciences, Wayne State Univ., Detroit, MI, 48202
Prostaglandins; 21(5):833-864 1981

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

17. GENITAL CANCER AND VIRUSES.
Rapp F, Jenkins FJ
Dept. Microbiology, Pennsylvania State Univ. Coll. Medicine, Hershey, PA, 17033
Gynecol Oncol; 12(2,part2):S25-S41 1981

18. VIRUS PAPILLOMAS IN HUMANS.
Nasemann T
Univ.-Hautklinik Eppendorf, Martinistr. 52, D-2000 Hamburg 20, W. Germany
Hautarzt; 32(11):601-606 1981
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DIETARY ASPECTS OF CARCINOGENESIS

SERIES CK03 No. 82/05

MAY, 1982

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STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. CARCINOGENICITY OF CARRAGEENAN.

Hopkins J
British Industrial Biological Res. Assoc.
Food Cosmet Toxicol; 19(6):779-781 1981

The carcinogenic potential of the food stabilizer carageenan is discussed. Carrageenan has been identified as a cocarcinogen in studies with rats, enhancing methylnitrosourea-, azoxymethane- and dimethylhydrazine-induced colonic carcinoma. Carageenan may also produce colonic carcinoma when used alone. (no Refs)

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
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2. COFFEE DRINKING AND CANCER OF THE LOWER URINARY TRACT.

Morrison AS, Buring JE, Verhoek WG, Aoki K, Leck I, Ohno Y, Obata K
Dept. Epidemiology, Harvard Univ. Sch. of Public Health, 677 Huntington Ave, Boston, MA, 02115
JNCI; 68(1):91-94 1982

The relation of coffee drinking to the incidence rate of cancer of the lower uri nary tract ('bladder cancer') was evaluated. Broadly based series of cases and series of controls drawn from the general population of each area were assembled and interviewed in Boston, Massachusetts (587 cases, 528 controls), Manchester, England (541 cases, 725 controls), and Nagoya, Japan (289 cases, 586 controls). Compared to drinkers of an average of less than one cup of coffee per day, those who drank more had a relative risk of bladder cancer estimated as 1.0 (0.8-1.2, 95% confidence interval). With adjustment for cigarette smoking habits, only small and irregular changes in risk were seen with increasing frequency of coffee consumption. Duration of coffee drinking showed little relation to risk of bladder cancer. Apparent associations of coffee drinking and bladder cancer may be due to incomplete control of the effect of cigarette smoking. If there is a true association of coffee drinking and bladder cancer, it is likely to be weak. (Author abstract) (12 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

3. EFFECT OF RETINOIDS ON THE INDUCTION OF COLON CANCER IN F344 RATS BY N-METHYL-N-NITROSOUREA OR BY 1,2-DIMETHYLHYDRAZINE.

Silverman J, Katayama S, Zelenakas K, Lauber J, Musser TK, Reddy M, Levenstein MJ, Weisburger JH
American Health Foundation, Naylor Dana Inst. Disease Prevention, Valhalla, NY, 10595
Carcinogenesis; 2(11):1167-1172 1981

The possible modifying effect of synthetic and natural retinoids on the incidence of colon cancer in rats induced by two intrarectal doses of 2.5 mg of N-methyl-N-nitrosourea (MNU) given once a wk for 2 successive wk or a single 150 mg/kg body weight dose of 1,2-dimethylhydrazine (DMH), sc was investigated. Emphasis was on the effect of the development of early tumors as visualized by endoscopy. With the retinoids N-ethyl-retinamide, N-2-hydroxyethylretinamide, N-(4-hydroxyphenyl)-all-trans-retinamide (RAHA) and retinyl acetate (RA) administered orally after the carcinogens, significant differences in early developing tumors were not found. At histopathological examination of the tumors the RAHA + DMH group had significantly fewer adenomas per animal. The percentage of adenoma bearing rats was significantly lower in groups receiving RAHA + DMH or RA + DMH. However, food consumption was lower in rats consuming either RAHA or RA Retinyl palmitate (RP) and RAHA were administered intrarectally to MNU-induced rats either before or after the carcinogen. When administered before MNU, RP caused a significant increase in the percentage of tumor bearing animals and the av number of tumors per animal as visualized endoscopically. At histopathological examination, all retinoid groups except RAHA given after the carcinogen, produced significantly more adenomas per animal and a significantly greater adenoma incidence than did the control groups. Thus, in tw o systems, the oral administration of retinoids did not clearly inhibit the early or later stages of colon tumor development. Intrarectal infusion of two retinoids had no effect on colonic morphology but at histopathological examination of later stage tumors there was an enhanced adenoma response. (Author abstract) (36 Refs)

4. VITAMIN A STATUS AND ESOPHAGEAL CANCER RISK: EPIDEMIOLOGIC AND EXPERIMENTAL EVIDENCE FOR A POSITIVE ASSOCIATION.

van Rensburg SJ, Kruger EF, Louw ME, Du Plessis JP
Natl. Res. Inst. Nutritional Diseases, P. O. Box 70, Tygenberg 7505, South Africa
Nutr Rep Int; 24(6):1123-1131 1981

To evaluate the role of vitamin A in the etiology of esophageal cancer (EC), the occurrence of EC was studied in the Transkei territory (South Africa). A moderate incidence district (9.0/100,000) was used as a low risk region and a high incidence (50.5/ 100,000) district was used as the high risk region. The survey of the dietary intake and serum levels of vitamin A showed that deficient intake of vitamin A was 1-3x more common in the low risk groups. Elevated serum values were detected in 2-4 and 6-9 yr old children from high risk group. Experiments in BD IX rats showed that the incidence of methylbenzylnitrosamine-induced EC in animals who received a diet containing 30 mg/kg of vitamin A was 2x higher than that in the animals who received a diet containing 3 mg/kg of vitamin A. The rats who received vitamin A deficient diet were free of EC. (15 Refs)

5. RETINOIDS AND THE PREVENTION OF CERVICAL DYSPLASIAS.

Romney SL, Palan PR, Duttagupta C, Wassertheil-Smoller S, Wylie J, Miller G, Slagle NS, Lucido D
Dept. Gynecology/Obstetrics, Albert Einstein Coll. Medicine, 1300 Morris Park Ave., Bronx, NY, 10461
Am J Obstet Gynecol; 141(8):890-894 1981

Women with abnormal cytology were matched with normal control subjects for age, parity, ethnicity, and socioeconomic class and participated in a blind case-control study focused on the role of nutrition in cervical dysplasia. Sucrose gradient ultracentrifugation studies for determination of the presence and concentration of the binding proteins for retinol and retinoic acid were performed on colposcopic biopsy tissue specimens. The nutritional survey revealed statistically significant differences for vitamins A and C and beta carotene. Retinol binding protein was absent or minimally detectable and inversely related to the severity of the dysplasia. It is proposed that a double-blind clinical trial be conducted to evaluate whether retinoids may pharmacologically inhibit, arrest, or reverse cervical dysplasia. (Author abstract) (16 Refs)

6. STUDIES ON THE IN VITRO METABOLISM OF AFLATOXIN B1 AND G1 IN THE LIVER OF RATS FED AT VARIOUS LEVELS OF VITAMIN C.

Domngang F, Bassir O
Service de biochimie Universite de Yaounde, B.P. 812 Yaounde, Cameroun
Biochem Pharmacol; 30(24):3317-3322 1981

Male and female weanling rats, divided into three groups, were maintained for 30, 60 and 90 days on an experimental diet without vitamin C. The drinking water containing or devoid of vitamin C was offered each day. The first group had no vitamin C; the second group or control had normal vitamin C and

the third group had above-normal vitamin C in the water. Twenty-four hr after the last feeding, the animals were decapitated and the liver microsomes plus soluble fraction (9,000 g) were prepared. This preparation was used for O-demethylase and the hydroxylase assays of aflatoxins B1 and G1 (AFB1 and AFG1). Results from O-demethylation in both control and experimental groups of both sexes, showed that O-demethylase activity is directly related to ascorbic acid content of the water. But the hydroxylase activity in male and female animals maintained on the same diet showed only slight differences. In the male, there was an increase with time of hydroxylase activity with AFB1 up to 60 days and a decrease at 90 days. Hydroxylation assay revealed that in the female, metabolism of AFB1 increased at 30 days but decreased at 60 and 90 days. (Author abstract) (29 Refs)

7. MODIFICATION OF MAMMARY CARCINOGENESIS AND TISSUE PEROXIDATION BY SELENIUM DEFICIENCY AND DIETARY FAT.

Ip C
Dept. Breast Surgery and Breast Cancer Res. Unit, Roswell Park Memorial Inst., Buffalo, NY
Nutr Cancer; 2(3):136-142 1981

The independence of selenium (Se) and unsaturated fat intake as modifiers of the incidence of mammary tumors induced by 7,12-dimethylbenz(a)anthracene (DMBA) and the liability of mammary tissue to lipid peroxidation were studied. Female Sprague-Dawley rats were fed one of six synthetic diets (three different types of diets with and without Se supplementation) from weaning until termination of the experiment. The three basal diets contained different levels of tocopherol-stripped corn oil, 1%, 5%, and 25%. Mammary tumors were induced by intragastric administration of a 5 mg dose of DMBA at 50 days of age. Measurement of Se-dependent glutathione peroxidase activity in the rat liver was used to determine the functional status of Se at 0, 10, 20, and 30 days after the rats were given either the basal Se-deficient diet or the supplemented regimen. In rats receiving an adequate supplement of Se (0.1 ppm) tumor incidence increased proportional to the amount of corn oil in the diet. A deficiency of Se in the diet resulted in a marked enhancement in tumor incidence and yield in rats fed the 25% corn oil regimen. Glutathione peroxidase activity in the mammary gland was not influenced by dietary fat but was severely depressed in the absence of added Se. Lipid peroxidation in the mammary tissue was elevated with increasing levels of dietary fat in rats supplemented w ith 0.1 ppm of Se. The unsupplemented 25% fat group showed significant enhancement of tissue peroxidation when compared to their supplemented controls. Determination of peroxide in the synthetic diets demonstrated that corn oil when mixed with the other ingredients, auto-oxidized very quickly especially at room temperature. The amount of peroxide formed is proportional to the content of corn oil and to the length of storage. Se deprivation did not affect the tumor growth rate or the proliferation of malignant vs benign lesions. (40 Refs)

8. ENHANCEMENT OF EXPERIMENTAL PANCREATIC CANCER IN SYRIAN GOLDEN HAMSTERS BY DIETARY FAT.

Birt DF, Salmasi S, Pour PM
Eppley Inst. for Res. in Cancer, Univ. Nebraska Medical Center, 42d and Dewey Ave., Omaha, NE, 68105
JNCI; 67(6):1327-1332 1981

The effects of dietary fat on the induction and development of pancreatic ductular adenocarcinoma were studied in random-bred Syrian golden hamsters. Diets containing low-fat (LF) or high-fat (HF) levels of corn oil [4.5 or 18.0 g/385 kilocalorie (kcal)], contributing 10 or 41% of the cal, respectively, were fed either before or after a single injection of N-nitrosobis(2-oxopropyl)amine (BOP) (10 mg/kg body wt). Control hamsters were fed corn oil at a medium-fat (MF) level (9 g/385 kcal) for life. The incidence of ductular adenocarcinomas increased in both males and females (LF diet, 16%; HF diet, 34%) when the HF diet was fed after BOP treatment. The av number of carcinomas per carcinoma-bearing animal also increased (LF diet, 1.3; HF diet, 3.0), but the carcinoma incidence was not influenced by these diets being fed before carcinogen treatment. The incidence of ductular adenomas was high with all treatments and was not influenced by diet. However, the number of adenomas was increased in animals fed HF diets. In addition, the incidence of acinar cell nodules was elevated in animals fed the MF and HF diets after BOP administration. These results showed that dietary fat modified the development of experimental ductular adenocarcinoma of the pancreas. (Author abstract) (31 Refs)

9. SIALIC ACID METABOLISM IN NUTRITIONALLY MODULATED MAMMARY GLAND CARCINOGENESIS.

Fox OF, Kishore GS, Carubelli R
Biomembrane Res. Lab., Oklahoma Medical Res. Foundation, 825 Northeast Thirteenth St., Oklahoma City, OK, 73104
Cell Mol Biol; 27(5):473-482 1981

The activities of CMP-N-acetylneuraminic acid synthetase, sialyltransferase and neuraminidase were investigated in female Sprague-Dawley rats with mammary adenocarcinomas induced by oral administration of 7,12-dimethylbenz[a]anthracene. The carcinogenic process was modulated by the levels and degree of unsaturation of the dietary lipids in the presence and in the absence of dietary antioxidants. The activities of the biosynthetic enzymes responsible for the activation and transfer of sialic acids, ie CMP-N-acetylneuraminic acid synthetase and sialyltransferase were higher in tumors than in control mammary glands. The cytosolic and membrane-bound neuraminidases, on the other hand, were higher in control tissue than in tumors. In the low-fat dietary group, which had the lowest rate of tumor incidence, a tocopherol effect on sialic acid metabolism was detected; rats fed low-fat diet supplemented with alpha-tocopherol exhibited the highest levels of control mammary gland sialyltransferase activity, whereas tocopherol deficiency was accompanied by a loss of the tumor-dependent enhancement of CMP-N-acetylneuraminic acid synthetase. The enzyme profiles in the livers of control and tumor-bearing rats, although less well defined, showed some of the general trends observed in mammary tissues. (Author abstract) (21 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

10. EPIDEMIOLOGY OF LYMPHOID MALIGNANCY. AN ETIOLOGICAL HYPOTHESIS OF ADULT T-CELL LEUKEMIA/LYMPHOMA.

Tajima K, Tominaga S
Dept. Epidemiology, Aichi Cancer Center Res. Inst., Chikusa-ku, Nagoya 464, Japan
Gan To Kagaku Ryoho; 8(7):1009-1016 1981

In order to study adult T-cell leukemia/lymphoma (ATLL), which is prevalent in Kyushu, an etiological hypothesis of ATLL is proposed. ATLL is found in limited zones in Kyushu, mainly in rural and coastal areas, which are warm in winter and humid in summer. The geographical distribution of filariasis which accompanies several lymphoreticular ailments and was prevalent until 20 yr ago, was found to be closely correlated with that of patients with ATLL. It was also suggested from analysis of the chronological changes in the av wt and height of school children by prefecture that the nutritional condition of the inhabitants of Kyushu might have been poorer than that of other districts, especially in the past. In these respects, it is conceivable that repeated exposure to filarial infection in association with undernourishment and some other factors might have played an important role in the etiology of ATLL. (30 Refs) (In JPN)

11. EPIDEMIOLOGY OF GASTRIC CANCER IN CHILE.

Armijo R, Detels R, Coulson AH
Div. Epidemiology, Public Health Sch., Univ. California, Los Angeles, CA
Bol Of Sanit Panam; 91(4):362-368 1981

After Japan, Chile has the highest gastric cancer mortality rate (GCM) in the world. GCM is lowest in northern and southern Chile (20/100,000) and highest in the middle region (Maule, 52.5/100,000). Levels of nitrates and nitrites have been studied in vegetables, saliva, and urine of children in different parts of Chile. (9 Refs) (In SPA)

12. ESOPHAGEAL CANCER AMONG BLACK MEN IN WASHINGTON, D.C. II. ROLE OF NUTRITION.

Ziegler RG, Morris LE, Blot WJ, Pottern LM, Hoover R, Fraumeni JF
Environmental Epidemiology Branch, Div. Cancer Cause and Prevention, NCI, NIH, P ublic Health Service, U.S. Dept. Health and Human Services, Bethesda, MD, 20205
JNCI; 67(6):1199-1206 1981

A case-control study of esophageal cancer was conducted among the black male residents of Washington, D.C., to find reasons for the exceptionally high risk in this population. The next of kin of 120 esophageal cancer cases who died during 1975-1977 and of 250 D.C. black males who died of other causes were interviewed. Five indicators of general nutritional status - fresh or frozen meat and fish consumption, dairy product and egg consumption, fruit and vegetable consumption, relative wt (wt/ht(2)), and number of meals eaten per day - were each significantly and inversely correlated with the relative risk of esophageal cancer. Associations with other food groups were not apparent. The least nourished third of the study population, defined by any of these five measures, was at twice the risk of the most nourished third. None of these associations was markedly reduced by controlling for ethanol consumption, the other major risk factor in this population; smoking; socioeconomic status; or the other nutrition measures. When the three food group consumption measures were combined into a single overall index of general nutritional status, the relative risk of esophageal cancer between extremes was 14. Estimates of the intake of vitamin A, carotene, vitamin C, thiamine, and riboflavin were inversely associated with relative risk; but each micronutrient index was less strongly associated with risk than were the broad food groups that provide most of the micronutrient. Thus no specific micronutrient deficiency was identified. Instead, generally poor nutrition was the major dietary predictor of risk and may partially explain the susceptibility of urban black men to esophageal cancer. (Author abstract) (31 Refs)

13. NUTRIENT INTAKES AMONG RURAL TRANSKEIANS AT RISK FOR OESOPHAGEAL CANCER.

Groenewald G, Langenhoven ML, Beyers MJ, Du Plessis JP, Ferreira JJ, Van Rensburg SJ
(c/o van Rensburg), Natl. Res. Inst. Nutritional Diseases, P.O. Box 70, Tygerberg 7505, South Africa
S Afr Med J; 60(25):964-967 1981

The frequency of low nutrient intakes was investigated in areas of moderate and high esophageal cancer incidence in Transkei by means of 24-hr recall and habitual intake studies in children and nursing mothers. The results in moderate- and high-risk regions were similar and habitual intakes of protein, energy, phosphorus, iron, thiamine and vitamin A were generally satisfactory. Intakes of less than two-thirds the Recommended Dietary Allowances (RDA) had a high frequency of inadequate intakes for calcium (90%), nicotinic acid (79%), riboflavin (55%) and ascorbic acid (50%). It is concluded that the possibility of long-standing deficiencies of the latter three vitaminsol consumption, and smoking
habits of 147 white male patients diagnosed with cancer of the esophagus were compared with the reports of 264 white male patients of comparable ages with diagnoses other than cancer. The associations of cigarette and alcohol use as risk factors were examined. Beer drinking was the beverage most associated with risk elevation. Persons reporting their occupation as laborers, service personnel, and operatives were considered as having significantly elevated relative risk (RR). RR associated with levels of vitamin A intake ranged from 0.68-1.28. Vitamin C intake was more highly associated with disease status. Because both vitamin A and vitamin C intake are highly dependent on the fruit and vegetable consumption habits of patients, the association of the simple reported frequency of consumption of fruit and vegetables was examined. RR ranging from 0.52-2.47 indicate a 4.75-fold increase in RR when consumption of fruits and vegetables was less than 40x/mo and was compared to a reported intake of greater than 81x/mo. The increase in risk represents a gradient consistent with a hypothetical dose-response relationship. No significant modification of the effect was observed across smoking, drinking, or smoking and drinking specific categories. No significant modification of the effect was observed in analyses of high risk groups of laborers, service and operative workers and other occupations, suggesting that the effects of diet may be independent of social class as measured by type of occupation. The findings of this investigation are consistent with evidence of lower risk associated with vegetable consumption in instances of colon, lung,

bladder, oral, and laryngeal cancers, and with evidence for animals of tumor inhibition by vegetable properties. (18 Refs)

15. CASE-CONTROL STUDY OF 100 CASES OF GASTRIC CANCER.

Ye E, Tao Z, Shi K, Qiu X, Xu W, Li J, Zhu Z, Wang Y, Cai H, Yu Q, Peng Y, Guo X
Dept. Hygiene, Shanghai Second Medical Coll., Shanghai, People's Republic of China
Zhonghua Yufangyixue Zazhi; 15(2):107-109 1981

Etiologic factors are described and analyzed for 100 cases of gastric cancer (72 men, 28 women; median age 60.67) and 100 cases of nongastric cancer from eight hospitals around Shanghai. Results of the analysis showed that persons who were born in Shanghai or who had contact with petroleum or petroleum products had a high risk of gastric cancer. There was a significant sex difference in the relationship between gastric cancer and some of its etiologic factors. Thus, habitual overeating, irregular m eals, and a history of stomach disease were the possible etiologic factors in males, while family history of tumors and habitual eating when angry were possible etiologic factors in females. No relationship was found between gastric cancer and smoked or fried foods or foods with high salt contents, as reported in the literature. Also, no significant difference was found between the gastric cancer group and the control group or between the sexes, which could be related to blood type (A, B, O), smoking, alcohol drinking, tea drinking, or neurologic factors. However, the rate of habitual ingestion of fresh vegetables was lower in the cancer group than in the control group. (no Refs) (.n CHI)

16. THE LOW INCIDENCE OF COLORECTAL CANCER IN A 'HIGH-RISK' POPULATION: ITS CORRELATION WITH DIETARY HABITS.

Rozen P, Hellerstein SM, Horwitz C
Ichilov Hosp., Weizmann St., Tel Aviv, Israel
Cancer; 48(12):2692-2695 1981

In Israel, the incidence of colorectal cancer is highest in European-American-born Jews, although this incidence is lower than that found in European-born Jews living in the United States. A significantly lower-than-expected incidence was found in a specific kibbutz established by immigrants of European origin. Matched population samples from this kibbutz and the city of Tel Aviv revealed significant differences in dietary habits. The kibbutz members ate more unavailable carbohydrate and ascorbic acid and had a higher dietary polyunsaturated to saturated fat (P/S) ratio than the city dwellers. Both groups ate more crude fiber, less saturated fat, and less beef than has been reported from America and European countries. This kibbutz diet seems to have prevented large bowel cancer, parallels the 'prudent' diet of Wynder, and possibly indicates the feasibility of colorectal cancer prevention. (Author abstract) (34 Refs)

17. EFFECTS OF DIETARY BRAN AND THE COLON CARCINOGEN 1,2-DIMETHYLHYDRAZINE ON A FECAL BETA-GLUCURONIDASE ACTIVITY IN MICE.

London JF, Clapp NK, Henke MA
Oak Ridge Graduate Sch. Biomedical Sciences, Univ. Tennessee, Oak Ridge, TN, 37830
Food Cosmet Toxicol; 19(6):707-711 1981

Daily variations in fecal beta-glucuronidase (BGLU) activity in BALB/c mice and variations in BGLU activity induced by different dietary brans and/or 1,2-dimethylhydrazine (DMH) were investigated. The effect of DMH on BGLU was significant. Three mo after DMH treatments, mice had persistently depressed BGLU activity compared to mice not treated with DMH. BGLU activities of animals on control or wheat bran diets were 4-5x higher than the activities in animals on either soya-bean bran or corn-bran diets and differed significantly from them. Animals fed the corn-bran diet consistently had the lowest BGLU activities in fecal stream. No synergistic action of any combination of variables was noted. (63 Refs)

18. DIETARY FIBER: IS IT REALLY HELPFUL?

Burkitt DP
Unit Geographical Pathology, St. Thomas's Hosp., London SE1, England
Geriatrics; 37(1):119-122,126 1982

Fiber in the diet is discussed; topics include defects of Western diet, cancer, diabetes, gallstones, obesity, ischemic heart disease, and history of disease. Culturally determined dietary patterns appear to be responsible for the high prevalence in Western societies of major diseases such as colon cancer, atherosclerotic heart disease, and diabetes. Five fundamental dietary changes that distinguish modern Western diets from those of the Third World. are reduction in consumption of carbohydrate, particularly starch, increased consumption of sugar, increased consumption of fat, increased consumption of salt, and reduced consumption of fiber and replacement of fiber derived from cereals, legumes, and tubers with that from fruits and leafy vegetables. Cereal fiber has a much greater influence on bowel content and behavior than that from fruits and leafy vegetables. Fiber has important mechanical and metabolic functions in addition to influencing the bacterial flora in the large bowel. Fiber-rich diets are believed to protect against large-bowel cancer and may provide some general protection against all diet-related cancers. The role of dietary fiber in other diseases is reviewed. Diets should include more starch, less sugar, less fat, more fiber, and less salt. (10 Refs)

PUBLIC POLICY CONCERNING DIETARY ASPECTS OF CARCINOGENESIS

19. REDUCTION OF HUMAN EXPOSURE TO ENVIRONMENTAL N-NITROSO COMPOUNDS.

Preussmann R, Spiegelhalder B, Eisenbrand G
Inst. Toxicology and Chemotherapy, German Cancer Res. Center, Im Neuenheimer Feld 280, 6900 Heidelberg, W. Germany
ACS Symp Ser; 174():217-228 1981

The complex pattern of human exposure to environmental N-nitroso compounds is summarized. Recent results in three areas (N-nitrosodimethylamine in beer; volatile N-nitrosoamines in baby nipples and pacifiers; occupational exposure in the rubber industry) where significant reductions of human exposure have been achieved are presented. Of the total daily intake of N-nitrosodimethylamine (NDMA) 0.7 ug/day results from the consumption of beer. NDMA is formed during kilning of green malt. Reduction of NDMA in malt was achieved by sulfuring during the first 10 hr. Nitrosamine contamination of rubber products originates from amine-containing accelerators and stabilizers used in the vulcanization process. In W. Germany a regulation is being discussed allowing not more than 10 ppb of preformed nitrosamines and 200 ppb of nitrosatable amines in migrates of nipples and pacifiers. In a tire fac-

tory nitrosodiphenylamine was substituted by cyclohexylthiophthalimid to remove the major nitrosating agent. Peroxides as accelerators can be used to solve the nitrosamine problem. (5 Refs)

20. CHEMICAL COMPOUNDS USED IN FOOD-PRODUCING ANIMALS; CRITERIA AND PROCEDURES FOR EVALUATING ASSAYS FOR CARCINOGENIC RESIDUES.

Food and Drug Administration
(c/o G. Guest) Bureau Veterinary Medicine, Dept. Health and Human Services, FDA, 5600 Fishers Lane, Rockville, MD, 20857
Fed Regist; 47(8,Book2):1767-1769 1982

A proposal is made for providing uniform criteria and procedures for evaluating the carcinogenic risk presented by residues of carcinogenic compounds used in food-producing animals. The selected approach would offer the fullest degree of public health protection by establishing an acceptable risk of carcinogenesis to humans and by taking into account the carcinogenic potency of drugs in extrapolating to the drug level in tissue which represents the acceptable level of risk. (no Refs)

GENERAL REVIEWS AND OTHER STUDIES

21. NUTRITIONAL ASPECTS OF CANCER PREVENTION: AN OVERVIEW.

Silverman J
American Health Foundation, Naylor Dana Inst Disease Prevention, Valhalla, NY, 10595
J Am Vet Med Assoc; 179(12):1404-1409 1981

Human epidemiologic studies and supporting laboratory animal data on the relationship between nutrition and modification of cancer are reviewed. Carcinogenesis results from at least two steps: initiation and promotion. Diet, a major risk factor, can influence cancer in several ways. Food additives or contaminants can act as initiators, promoters, or inhibitors; nutritional deficiencies can cause biochemical changes. Nutritional excesses can cause metabolic changes which may then promote or inhibit cancer. A decrease in caloric intake leads to an increase in tumor latency and a decrease in the number of tumors. The incidence of breast cancer in humans is positively correlated with the consumption of animal protein, fat, and simple carbohydrates. Dietary fat also exerts a promotional effect on colonic cancer. Cancer of the prostate and pancreas is positively correlated with fat consumption, but the evidence is not absolute. Vitamin A apparently inhibits tumor formation during the promotional phase. Vitamin C does not manifest a clear protective effect against any cancer. Studies with selenium have produced disparate results, while nitrosamines have shown carcinogenicity in many species. In most cases nutrients modify rather than cause cancer. (100 Refs)

22. N-NITROSO COMPOUNDS: DIET AND CANCER TRENDS; AN APPROACH TO THE PREVENTION OF GASTRIC CANCER.

Weisburger JH
American Health Foundation, Valhalla, NY, 10595
ACS Symp Ser; 1740:305-317 1981

Documented evidence for human cases of cancer unambiguously associated with short chain dialkylnitrosamines detected in foods in the Western world has not been presented yet. Other high risk regions have in common low intakes of foods with vitamin C. The risk factors for human gastric cancer include foods pickled with salt and saltpeter or residence in areas with high geochemical nitrate. Mutagenic activity was found for Salmonella typhimurium TA 1535 with an extract of a pickled fish frequently eaten in high risk Japan, which also induced glandular stomach cancer in rats. The formation of the mutagen can be blocked by vitamin C. In the control group (8 rats) fish extract alone was given, and no neoplastic changes in the stomach or pancreas were seen. Twelve rats were administered with extract of nitrite-treated fish. Glandular hyperplasia (6/12), squamous cell hyperplasia of the forestomach epithelium (5/12), and intestinal metaplasia was noted in this group. Several other neoplasias were noted in this group. Formation of nitrosoureido derivative can be blocked by vitamin C, vitamin E, and some other substances such as gallates. (66 Refs)

23. AN EPIDEMIOLOGICAL BASIS FOR CANCER CONTROL.

Armstrong B
NH and MRC Res. Unit in Epidemiology and Preventive Medicine, Univ. Western Australia, Dept. Medicine, Nedlands, Australia
Cancer Forum; 5(3):136-143 1981

Primary and secondary prevention of cancer, particularly cancer of the lung, breast, and colon and melanoma, which together account for greater than 40% of new cancer cases and premature mortality is discussed. Primary prevention aims at preventing cancer from occuring by ending exposure to the causal agent either by environmental or life type control. Lung cancer is attributable mainly to cigarette smoking; some cases result from occupational exposure. Breast cancer is associated with nulliparity, first full-term pregnancy at a comparatively late age, low parity, diet, radiation, post-menopausal estrogens, and oral contraceptives. Colon and rectal cancer are mainly related to dietary fiber deficiency and excess fat and meat consumption. Melanoma of the skin is due to exposure to UV radiation. Etiologies of other neoplasms are discussed: brain neoplasms (phenobarbitone use), prostate cancer (diet, cadmium exposure), stomach cancer (diet), leukemia (x-rays, benzene exposure, antineoplastic agent use), pancreatic cancer (diet, cigarette smoking), bladder cancer (occupational exposure to aromatic amines, cigarette smoking), and cervix cancer (sexual promiscuity). Dietary modification, cessation of smoking, and avoidance of excessive exposure to UV light was recommended. Screening methods involved in the secondary prevention of cancer include cervical cytology, proctosigmoidoscopy, and mammography at regular intervals for certain segments of the population. The choice between primary and secondary prevention of cancer is discussed. (20 Refs)

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As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. ETHYLENE OXIDE (ETO): EVIDENCE OF CARCINOGENICITY.
Coene RF
National Inst. for Occupational Safety and Health, Cincinnati, OH
Vet Hum Toxicol; 23(6):439-443 1981

DIETARY FACTORS IN CARCINOGENESIS

2. NUTRITIONAL ASPECTS OF CANCER PREVENTION: AN OVERVIEW.
Silverman J
American Health Foundation, Naylor Dana Inst Disease Prevention, Valhalla, NY, 10595
J Am Vet Med Assoc; 179(12):1404-1409 1981

3. FORMATION OF N-NITROSO COMPOUNDS IN FOODS.
Gray JI
Dept. Food Science and Human Nutrition, Michigan State Univ., East Lansing, MI, 48824
ACS Symp Ser; 174():165-180 1981

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

4. LYMPHOMAS AND LEUKAEMIAS. PART 2: TROPICAL AMERICA.
Jimenez E
Hospital Nacional de Ninos 'Dr. Carlos Saenz Herrera', Apartado 1654, San Jose, Costa Rica
Clin Haematol; 10(3):894-915 1981

5. LYMPHOMAS AND LEUKAEMIAS. PART 1: TROPICAL AFRICA.
Olweny CL
Uganda Cancer Inst., P.O. Box 3935, Kampala, Uganda
Clin Haematol; 10(3):873-893 1981

6. MALIGNANT MELANOMA.
Creagan ET
Div. Medical Oncology, Mayo Clinic, Rochester, MN, 55905
Compr Ther; 7(12):36-44 1981

7. MELANOTIC TUMORS OF THE UVEA AND RETINA.
Schmitt H
Universitats-Augenklinik, Theodor-Stern-Kai 7, D-6000 Frankfurt am Main, W. Germany
Med Klin; 76(24):672-675 1981
In GER

CELLULAR AND MOLECULAR MECHANISMS IN CARCINOGENESIS

8. THE NATURAL HISTORY OF CARCINOGENESIS: IMPLICATIONS OF EXPERIMENTAL CARCINOGENESIS IN THE GENESIS OF HUMAN CANCER.
Pitot HC, Goldsworthy T, Moran S
McArdle Lab. for Cancer Res., Dept. Oncology, Medical Sch., Univ. Wisconsin, Madison, WI, 53706
J Supramol Struct Cell Biochem; 17(2):133-146 1981

9. CURRENT CONCEPTS AND CONTROVERSIES IN CHEMICAL CARCINOGENESIS.
Weinstein IB
Div. Environmental Sciences, Inst. Cancer Res., Columbia Univ. Coll. Physicians and Surgeons, New York, NY, 10032
J Supramol Struct Cell Biochem; 17(2):99-120 1981

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

10. BURKITT'S LYMPHOMA.
Ambrus JL, Ambrus CM
Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263
J Med; 12(6):385-413 1981

11. PRIMARY HEPATIC CARCINOMA.
Iosquin CJ
Catedra de Medicina Interna A, Facultad de Ciencas Medicas, La Plata, Argentina
Rev Fac Cienc Med La Plata; 4(2):27-31 1981
In SPA

METHODOLOGY IN CARCINOGENESIS RESEARCH

12. MUTAGENESIS AND CARCINOGENESIS TESTING IN VITRO.
Casto BC
Northrop Services, Incorporated, P O. Box 12313, Research Triangle Park, NC, 27709
Biotechnol Bioeng; 23(12):2659-2671 1981

OTHER REVIEWS IN CARCINOGENESIS

13. CANCER IN THE YEAR 2000. A REVIEW.
Saxen E
Suomen Syoparekisteri, Liisankatu 21 B, 00170 Helsinki 17, Finland
Duodecim; 97(20):1708-1716 1981
In FIN

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SERIES CK03 No. 82/06 JUNE, 1982

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- GENERAL REVIEWS AND OTHER STUDIES..... 28-31

STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. WITHDRAWAL TIME REQUIRED FOR CLEARANCE OF AFLATOXINS FROM PIG TISSUES.

Furtado RM, Pearson AM, Hogberg MG, Miller ER, Gray JI, Aust SD

Dept. Food Science and Human Nutrition, Michigan State Univ., East Lansing, MI, 48824

J Agric Food Chem; 30(1):101-106 1982

Pigs were maintained on aflatoxin-spiked diets for 42 days and analyzed for tissue clearance of toxins. Pigs were found to rapidly metabolize and clear their tissues of aflatoxins once placed on an uncontaminated diet. This supports the suggestion that the risk from eating liver or meat of animals fed an aflatoxin-contaminated diet are negligible. (31 Refs)

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2. TRANSMISSION AND DISTRIBUTION OF AFLATOXIN IN CONTAMINATED BEEF LIVER AND OTHER TISSUES.

Stubblefield RD, Shotwell OL, Richard JL, Pier AC
Northern Regional Res. Center, Agricultural Res., Science and Education Admin., Peoria, IL, 61604
J Am Oil Chem Soc; 58(12):1015A-1017A 1981

Distribution of aflatoxin in the liver of a steer fed 52 mg of aflatoxin B1 equivalents/day for 5 consecutive days, and steps necessary to obtain a representative assay sample, were studied. Concentrations of various aflatoxins (B1, B2, G1, G2 and M1) were determined in the beef liver and bovine tissues. Degradation of aflatoxin M1 occurred even in the frozen state, indicating aflatoxin analysis should be performed as quickly as possible. (12 Refs)

3. EXPERIMENTAL STUDIES OF LUNG ADENOCARCINOMA IN MICE INDUCED BY CORN FLOUR INOCULATED WITH ASPERGILLUS FLAVUS.

Wang F, Zhang Z, Jiao R, Wang Z
Hebei Medical Coll., Shijiazhuang, People's Republic of China
Zhonghua Zhongliu Zazhi; 3(2):91-93 1981

Although Aspergillus flavus (AF) was the major fungus isolated from contaminated foodstuffs in Hebei Province where there was a high incidence of esophageal cancer, experimental carcinogenesis studies using this fungus could not produce esophageal, gastric or liver cancer in animals. Instead, lung cancer was induced. Corn flour inoculated with AF was fed to 40 Kunming mice (20 each sex) for 322 days followed by regular feed until the time for pathological examination, (day 501 and 545). The control group consisted of 40 mice (20 each sex) fed regular feed. During the feeding period, natural death occurred in 7 mice of the study group and in 10 mice of the control group. At the end of this period, lung adenocarcinomas were observed in 45.5% (15/33) of the study group while none was found in the 30 surviving control mice. The adenocarcinomas could be classified into three types, namely, high columnar, cuboid, and poorly differentiated. Based on these findings, AF is considered an etiologic factor for human lung cancer. (12 Refs) (In CHI)

4. A NATIONAL SURVEY OF MYCOTOXINS IN CANADA.

Andrews RI, Thompson BK, Trenholm HL
Deloitte Haskins and Sells Associates, Calgary, Alberta T2P 3G6, Canada
J Am Oil Chem Soc; 58(12):989A-991A 1981

Data collected as part of national survey of mycotoxins and mycotoxicoses in Canada are presented. A survey of research institutions across Canada showed that the mycotoxins, zearalenone, ochratoxin A and aflatoxin, were detected in suspected feed samples and produced mycotoxicoses in poultry, hogs and cattle. Concern was expressed that mycotoxins may enter the food chain, either directly, in plant products, or indirectly, as residues in meat products. (28 Refs)

5. AFLATOXIN IN FRESHLY HARVESTED 1979 GEORGIA CORN AND FORMATION AFTER COLLECTION.

Shotwell OL, Kwolek WF, Hesseltine CW
Northern Regional Res. Center, United States Dept. Agriculture, Peoria, Il, 6160 4
J Am Oil Chem Soc; 58(12):980A-983A 1981

Results of a study are reported in which aflatoxin in freshly harvested 1979 Georgia dent corn was determined and toxin formation after collection was studied. Significant increase in aflatoxin positive samples was associated with direct shipment to Illinois before drying, indicating that aflatoxin formed during the av of 7 days required for the trip. (7 Refs)

6. VARIABILITY IN CORN HYBRID RESISTANCE TO PREHARVEST AFLATOXIN CONTAMINATION.

Lillehoj EB, Zuber MS
Southern Regional Res. Center, AR-SEA, USDA, 1100 Robert E. Lee Blvd., New Orleans, LA, 70179
J Am Oil Chem Soc; 58(12):970A-973A 1981

Studies on variability in corn hybrid resistance to preharvest aflatoxin contamination are reviewed and the techniques for reduction of intrinsic variability and acquiring definite information on hybrid differences in susceptibility to contamination presented. A basis for further characterization of a genetic facility for resistance to the toxin-producing fungi is provided. (15 Refs)

7. REDUCING AFLATOXIN CONTAMINATION IN PEANUT GENOTYPES BY SELECTION AND BREEDING.

Mixon AC
Coastal Plain Station, Univ. Georgia, Tifton, GA, 31793
J Am Oil Chem Soc; 58(12):961A-966A 1981

Development of peanut genotypes resistant to seed colonization by aflatoxin-producing strains of Aspergillus species is explored. Factors associated with such resistance in the peanut include: cell structure and arrangement, permeability, waxy surface, tannin content and amino acid content of the seed testae. (68 Refs)

8. ASPERGILLUS FLAVUS CONTAMINATION OF NUTS, ALMONDS, AND CORN WITH KNOWN AFLATOXIN CONTENT.

Blaser P, Schmidt-Lorenz W
Institut fur Lebensmittelwissenschaft, Eidg. Technischen Hochschule, Laboratorium fur Lebensmittel-Mikrobiologie, 8092 Zurich, Switzerland
Lebensm Wiss Technol; 14(5):252-259 1981

Aflatoxins content and the quantity of colony forming units (CFUs) of Aspergillus flavus were determined on 154 samples of nuts and corn. No correlation were found between aflatoxin content of the samples and number of CFUs or between aflatoxin content of the samples and aflatoxin production by isolated Aspergillus colonies. Of 218 isolated colonies examined, 27.5% produced aflatoxin B1, 13.5% produced aflatoxin B2, 11.5% produced aflatoxin G1, and 4.5% produced aflatoxin G2. (20 Refs).(In GER)

9. THE AFLATOXIN SITUATION IN DIFFERENT KINDS OF FOODSTUFFS.

Woller R, Majerus P
Chemisches Untersuchungsamt Trier, Maximinerstr 11a, D-5500 Trier, W. Germany
Mitt Geb Lebensmittelunters Hyg; 72(4):437-440 1981

Amounts of aflatoxin (AT) in 441 commercial samples of hazel nuts, walnuts, grated coconut, poppy seeds, and sesame seeds were measured. No AT was found in coconut, poppy seeds, or sesame s eeds. AT was found in 2/185 hazel nut samples (16 and 106 ug/kg) and in one walnut sample (18 ug/kg). All three samples containing AT were moldy. (12 Refs) (In GER)

10. THE PRESENCE OF AFLATOXIN AND SOME POLYCYCLIC AROMATIC HYDROCARBONS IN HUMAN FOODS.

Emerole GO, Uwaifo AO, Thabrew MI, Bababunmi EA
Biomembrane Res. Section, Dept. Biochemistry, Univ. Ibadan, Ibadan, Nigeria
Cancer Lett; 15(2):123-129 1982

Analysis of common food items sold in Nigerian markets demonstrated the presence of benzo(a)pyrene (BP) and benz(a)anthracene (BA) in all fish and meat samples and aflatoxin in the food crops and spices tested. The highest hydrocarbon levels were found in moonfish, which contained 7.5 ug/kg BA, and in smoked meat, which contained 15.5 ug/kg BA and 8.5 ug/kg BP. Manihot flour, millet, corn, and groundnuts contained 1.2-1.7 mg/kg aflatoxin, whereas rice contained only 0.04 mg/kg. Results are discussed in relation to the relatively high incidence of cancer in tropical Africa. (32 Refs)

11. IMPLICATIONS OF SEASONAL VARIATIONS IN AFLATOXIN B1 LEVELS IN NIGERIAN MARKET FOODS.

Okonkwo PO, Obionu CN
Dept. Pharmacology and Therapeutics, Faculty Medicine, Univ. Nigeria, Enugu, Nigeria
Nutr Cancer; 3(1):35-39 1981

Seasonal variations in the aflatoxin-B1 (AB1) levels in tropical foods are reported and ingestion patterns of AB1 are calculated for various communities. Cassava showed a highly significant difference in AB1 levels between the rainy and dry seasons. Three other items (peanuts, rice, and yams) also showed seasonal variations. The ratio of rainy season over dry season mean values for AB1 ranged from 2.80 to 1.65. Dried fish and palm oil showed no seasonal variations in AB1 levels. The risk to man from seasonal exposure to aflatoxins is discussed. (22 Refs)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

12. STUDIES TO EVALUATE ARTIFICIAL SWEETENERS, ESPECIALLY REMSEN-FAHLBERG SACCHARIN, AND THEIR POSSIBLE IMPURITIES, FOR POTENTIAL MUTAGENICITY BY THE SALMONELLA/MAMMALIAN LIVER MICROSOME TEST.

Herbold BA
Inst. Toxicology, Bayer AG, Postfach 101709, D-5600 Wuppertal 1, W. Germany
Mutat Res; 90(4):365-372 1981

Mutagenic activity (MA) of artificial sweeteners and some possible impurities of artificial sweeteners was studied in Salmonella typhimurium with or without metabolic activation by S9. S9 was prepared from livers of Sprague-Dawley rats. Bacterial cultures were exposed to saccharin (Sacc: less than or equal to 12,500 ug/plate), o-toluene sulfonamide or p-toluene sulfonamide (less than or equal to 18,000 ug/plate), o-sulfamoylbenzoic acid, p-sulfamoylbenzoic acid, o-sulfobenzoic acid, p-sulfobenzoic acid, phthalimide, anthranilic acid, disulfide salt, boiled saccharin, or one of three saccharin extracts (all less than or equal to 2,500 ug/plate), cyclamate (less than or equal to 12,500 ug/plate), or cyclohexylamine (less than or equal to 2,500 ug/plate). Cyclophosphamide (less than or equal to 100 ug/plate) and trypaflavine (less than or equal to 200 ug/plate) were used as positive controls. All Sacc tested was prepared by the Remsen-Fahlberg process. With the exception of one Sacc extract, all chemicals tested were negative for MA in the standard Ames assay. One organic extract of Sacc produced inconclusive results in three consecutive experiments. Results indicate that neither Sacc, cyclamate, cyclohexylamine, nor known impurities of the sweeteners are mutagenic in the Salmonella/mammalian liver microsome test. (23 Refs)

13. ARTIFICIAL SWEETENERS IN RELATION TO THE EPIDEMIOLOGY OF BLADDER CANCER.

Howe GR, Burch JD
NCI Canada Epidemiology Unit, Univ. Toronto, Ontario, Canada
Nutr Cancer; 2(4):213-216 1981

Three types of studies of the possible association between artificial sweeteners and cancer are reviewed; temporal trend studies, studies of diabetics, and case-control studies. Animal studies have provided strong evidence for the carcinogenicity of saccharin. The methodological problems need further investigation. Avoidance of the use of artificial sweeteners is advised. (23 Refs)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

14. MUTAGENIC EFFECT OF AN ANTHRAQUINONE ON HUMAN LYMPHOCYTES.

Carballo MA, D'Aquino M, Aranda EI
Centro Nacional de Genetica Medica, Combate de los Pozos 2193, 1245 Buenos Aires, Argentina
Medicina (B Aires); 41(5):531-534 1981

The effect of 1,8-dihydroxyanthraquinone in dimethylsulfoxide peripheral human lymphocytes was studied in vitro. A clastogenic effect was observed under the conditions employed. Synthetic and natural anthraquinones have wide distribution; they are used in food coloring, cosmetics, pharmaceuticals, and laxatives. (10 Refs) (In SPA)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

15. SERUM RETINOL AND THE INVERSE RELATIONSHIP BETWEEN SERUM CHOLESTEROL AND CANCER.

Kark JD, Smith AH, Hames CG
Dept. Social Medicine, Sch. Public Health and Community Medicine, Hebrew Univ., Ein Karem, Jerusalem, Israel
Br Med J; 284(6310):152-154 1982

Several human studies have shown an inverse relation between vitamin A intake (and serum concentrations of retinol and carotene) and cancer. Serum cholesterol concentrations have also been reported in inverse relation to cancer. In a study of 3,102 people in Evans County, Georgia, who were followed for over 12-14 yr to assess the incidence of cancer there was an inverse association between the risk of cancer and both serum retinol and serum cholesterol concentrations. The

data also showed an unexpectedly strong correlation between serum retinol and total cholesterol concentrations. The inverse relationship wit h cancer was stronger with serum retinol than with cholesterol, which suggested that the association with cholesterol might be secondary. This suggestion may also explain the cholesterol-cancer association reported in several other cohort studies. Further studies of the relation between serum concentrations of cholesterol, retinol, and carotene and the incidence of cancer are needed. (Author abstract) (34 Refs)

16. THE EFFECTS OF VITAMIN E ON MOUSE FITNESS AND SURVIVAL.

Blackett AD, Hall DA
Dept. Medicine, General Infirmary, Univ. Leeds, Leeds, England
Gerontology; 27(3):133-139 1981

A small colony of C3H/He and LAF1 mice, of which 50% were receiving a diet supplemented with vitamin E (0.25%, w/w, dl-alpha-tocopherol), was set up for investigation of the reported action of antioxidants on increasing longevity. It was found that vitamin E exerted no effect on maximum longevity, but increased the numbers of both genotypes surviving to 24 mo. The beneficial effects appeared to act by reducing the incidence of fatal tumors late in life and by counteracting a debilitating condition early in life. It is suggested that this debility may have resulted from low antioxidant in the control diet. The relevance of free radicals to ageing is questioned. (Author abstract) (21 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

17. EPIDEMIOLOGICAL ASSESSMENT OF DIETARY INTAKE.

James WP, Bingham SA, Cole TJ
Medical Res. Council's Dunn Clinical Nutrition Center, Adenbrooke's Hosp., Cambridge, England
Nutr Cancer; 2(4):203-212 1981

The validity of techniques for quantitating nutrient intake of individuals or populations being studied for factors involved in carcinogenesis, and the problems involved in current dietary studies, are reviewed. Epidemiological analyses of the relationship between diet and disease examine the dietary practice of individuals and groups of people over a period of years so that the risk of cancer is known. Food balance sheets, household surveys, and studies of specific groups (24-hr recall, dietary history, questionnaires, weighted food records) have uncertain validity. The suitability of weighted food intake records for individuals, although probably the most accurate method, is weakened by individual variability in food intake from day to day. Application of biological markers and nonradioactive isotopes could supply better information about the intake of vitamins and minerals monitored for their supposed role in preventing carcinogenesis. New techniques for rapid measurement of metabolic rate, based on a portable apparatus that samples expired air and integrates oxygen measurements with flow rates to give estimates of metabolic rate, are becoming available. Measurements of urinary N can also be used for assessing the food intake and accuracy of the obtained data about dietary habits of individuals. Biological markers of food intake can provide independent indicators of intake unaffected by the subject's poor memory for food ingested, his bias in reporting food consumed, or his tendency to alter dietary habits once these are being investigated. (53 Refs)

18. LACK OF EFFECT OF DIETARY FAT ON THE GROWTH AND ESTROGEN SENSITIVITY OF THE MT-W9B TRANSPLANTABLE MAMMARY TUMOR.

Ip MM, Ip C
Dept. Experimental Therapeutics, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263
Nutr Cancer; 3(1):27-34 1981

Effects of diets containing low- (LF: 0.5%) or high (HF: 20%) fat on the growth and estrogen responsiveness of the MT-W9B mammary tumor in Wistar-Furth rats were studied. Tumor growth was not significantly affected by HF or LF diet. Tumor growth was also unaffected in either group by ovariectomy (OVx), sham OVx, or by OVx with estradiol supplementation. Dietary fat intake did not alter the sensitivity of the uterus to estrogen. Progesterone receptor (PR) levels were slightly elevated in tumors from sham-OVx rats fed a HF diet compared those fed a LF diet; this increase was not statistically significant. Responses of PR to OVx or to OVx with estrogen supplementation were not altered by dietary fat levels. PR levels were decreased in OVx rats to 16% and 14% of control levels in LF and HF groups, respectively, and increased 16-fold and 13-fold, respectively, in OVx rats given estradiol. In the same tumors, estrogen receptor levels were not significantly altered by either dietary or hormonal manipulation. PR levels were elevated in uteri from sham-OVx rats fed a HF diet compared to those fed a LF diet when expressed on a picomole/uterus basis, but this increase was not statistically significant when expressed on a per mg basis. Dietary fat did not appear to affect the response of uterine PR to estrogen manipulation. Dietary effects on estrogen receptors are described. The results suggested that dietary fat does not influence the sensitivity of normal or neoplastic tissue to estrogen action. (37 Refs)

19. INFLUENCE OF DIET ON MAMMARY CANCER.

Carroll KK
Dept. Biochemistry, Univ. Western Ontario, London, Ontario, N6A 5C1, Canada
Nutr Cancer; 2(4):232-236 1981

Analysis of epidemiolcgical data and experiments with animals have provided evidence that dietary fat has an important influence on mammary cancer. Epidemiological data on dietary fat in relation to breast cancer, mechanisms of action of dietary fat in mammary carcinogenesis, and effects of dietary components, other than fat, on mammary carcinogenesis are reviewed. The epidemiological data indicate that total dietary fat shows a better correlation with breast cancer mortality than either animal fat or fat derived from plant sources. More information on the relationship between diet and breast cancer will enable a better assessment of the potential value of the dietary approach to prevention or treatment. Restriction of calories from fat has value not only as a possible means of reducing cancer incidence, but also in decreasing the incidence of heart disease and obesity. (44 Refs)

20. IS COLON CANCER A DISEASE OF CIVILIZATION?.

Raiser MW
Div. Gastroenterology, Edgewater Hosp., 5700 N. Ashland Ave., Chicago, IL, 60660
Minerva Dietol Gastroenterol; 27(4):638-640 1981

Epidemiology of carcinoma of the colon (CC) is discussed. CC has become increasingly common in North America and Western Europe in the last 30 yr. These areas have higher income, a higher standard of living, and higher meat consump-

tion. A diet rich in meat and associated animal fat increases the production of cholic acids. Feces have higher amounts of cholic acids and their degradation products, some of which are carcinogens. Constipation, another problem associated with a higher standard of living, increases the amount of time feces are in contact with intestinal walls. Japanese, Poles, and other Eastern Europeans, all from areas that are low in CC, become as prone to CC as the rest of the population after emigration to the United States. On the other hand, Seven Day Adventists living in Loma Linda, California and consuming vegetarian diets have a much lower CC rate than the rest of the population; the amount of cholic acids and their derivatives in their feces is much lower. Another factor associated with diet is the type of bacterial flora and enzymes active in the intestines (beta-glucuronidase, nitroreductase, azoreductase etc). Associated with these problems is increased intestinal diverticulosis. Current dietary recommendations are for decreased total calories, animal fat, and cholesterol, and increased vegetable fibers and unsaturated fats. (14 Refs) (In ITA)

21. SHOULD WE EAT MORE FIBRE?

Stephen AM
Dunn Clinical Nutrition Centre, Addenbrookes Hosp., Trumpington St., Cambridge CB2 1QE, England
J Hum Nutr; 35(6):403-414 1981

Various aspects of the role of dietary fiber in health are discussed: epidemiological studies, effects of fiber on fecal weight, diverticular disease, colon cancer, ischemic heart disease, and the possibility of the recommendation of a standard fiber intake. Outline of a diet fed to volunteers, containing 40 g dietary fiber/day, is presented. The type of fiber to be recommended is discussed. (62 Refs)

22. COFFEE CONSUMPTION AND PANCREATIC CANCER: TEMPORAL AND SPATIAL CORRELATION.

Benarde MA, Weiss W
Dept. Community Medicine and Environmental Health, Hahnemann Univ. Health Sciences, Philadelphia, PA, 19102
Br Med J; 284(6313):400-402 1982

An examination of available data on per capita consumption of coffee and pancreatic cancer mortality in the United States since 1950 shows a temporal association. A rise and fall in coffee consumption was followed by a rise and fall in the incidence of pancreatic cancer with roughly a 10-yr lag. Nevertheless, there were inconsistencies in this relationship by sex and race. An unimpressive spatial relationship was also found between the consumption of coffee and pancreatic cancer mortality in 13 countries. While this relationship suggests an association, major inconsistencies cast doubt on the possibilities that it is one of cause and effect. This may be due to confounding, particularly by cigarette smoking. The association between cigarette smoking and cancer of the pancreas is much more consistent with a causal relationship. (Author abstract) (25 Refs)

23. DIET AND CANCER OF THE LARGE BOWEL.

Zaridze DG
Analytical Epidemiology Programme, Div. Epidemiology and Biostatistics, International Agency for Res. on Cancer, 150 Cours Albert Thomas, 69372 Lyon, Cedex 2, France
Nutr Cancer; 2(4):241-249 1981

Suspected etiological and protective factors in large bowel cancer are reviewed. Incidence of large bowel cancer correlates with consumption of fat, protein or meat. Etiological hypotheses propose that high-fat diet is related to an increased risk of large bowel cancer, while analysis of the geographical distribution of colon cancer in humans. The results of further epidemiological studies do not unequivocally support the hypotheses. Control studies do not confirm an association between diet and the colorectal cancer risk at the individual level. Lack of uniformity among the findings of epidemiological studies concerned with the role of diet could be explained by lack of sensitive and reproducible methods for assessment of diet in epidemiological studies and lack of knowledge of the mode of action of dietary factors responsible either for the development of the tumor or for protection against it. Research on large bowel cancer in humans is focused on the promoting factors. Initiators could be the factors responsible for the individual risk for colon cancer in populations with homogeneous high-fat diet practices. Available data indicate adenomatous polyps and non-invasive carcinoma of the large bowel could remain stationary. Future epidemiological research on diet and large bowel cancer should deal with more precisely defined hypotheses in which the multifactorial nature of the tumor would be taken into account. (49 Refs)

24. AN EPIDEMIOLOGICAL STUDY ON THE POSSIBLE ASSOCIATION BETWEEN INTAKE OF BROILED FISH AND CANCER (MEETING ABSTRACT).

Ikeda M, Yoshimoto K, Yoshimura T, Kono S, Kato H, Kuratsune M
Department of Public Health, Fac. of Medicine, Fukuoka, Japan
Proceedings of the Japanese Cancer Association, 40th Annual Meeting, October 1981, Sapporo, Japan. The Association 415 pp., 1981.

Yr ago, the Radiation Effects Research Foundation investigated 7,553 subjects for dietary habits such as frequency of consumption of broiled fish, milk, and others. Therefore, the possible association between these habits and their cancer deaths seen during 11 yr was investigated by the linear multiple stepwise regression analysis. Frequent consumption of broiled fish was significantly positively associated with deaths from all sites of cancer and those from gastric cancer. Liver cancer was also associated with this habit but with no statistical significance. (no Refs)

25. NUTRITION AND GASTRIC CANCER.

Joossens JV, Geboers J
33, Capucijnenvoer, B-3000 Leuven, Belgium
Nutr Cancer; 2(4):250-261 1981

Statistical methods and data from different countries were used to study the cause of gastric cancer (GC) and stroke mortality. GC has a multifactorial origin. Possible causes include genetic factors, soil, and nutritional and socioeconomic factors. GC mortality rates are decreasing in 23 of 26 countries studied. Results of multiple regression analysis of the GC-stroke mortality relationship are presented. A strong association was observed for each sex separately and for both sexes combined. Lung cancer is also related to stroke mortality. According to a working hypothesis set forth in 1965, salt intake is the predominant linking factor for both GC and stroke mortality. Thus, stroke would be influenced by the relationship between salt and blood pressure; GC would result from the delaying and caustic properties of stomach hypertonicity, possibly producing atrophic gastritis. Atrophic gastritis favors the synthesis of endogenous nitrites and of nitroso-carcinogens. The salt hypothesis offers an explanation for the geographical and time behavior of GC and stroke mortality, and the socioeconomic gradient of both diseases. GC mortality can also be used to provide a rough estimate of the salt intake in a given country and yr. The influence of salt was tested in Belgium through a delib-

erate attempt to lower the salt intake of the population. The observed decreases in GC and stroke mortality were consistent with the measured decrease in salt excretion. Monitoring 24-hr salt and creatinine excretion in different countries and over a longer period of time is needed. (72 Refs)

26. RELATION BETWEEN CANCER DEATH AND FOODS CONSUMPTION --- SEVEN SITES IN DIGESTIVE ORGANS (MEETING ABSTRACT).

Nagai M, Hara N, Sakata K, Hashimoto T, Yanagawa H
Department of Public Health, Jichi Medical School, Tochigi, Japan
Proceedings of the Japanese Cancer Association, 40th Annual Meeting, October 1981, Sapporo, Japan. The Association 415 pp., 1981.

The areal difference of the mortality from cancer of 7 sites of digestive organs and consumption of foods in 46 prefectures and their capital cities were observed and analyzed by correlation and factor analysis. The standardized mortality ratios (SMR) for male were calculated over 10 yr (1969-'78). The variables on food consumption were selected from two surveys conducted by Prime Minister's Office. The foods statistically suspected as risk factors are: 1) pork, oil and shochu for esophagus cancer; 2) fresh fishes, salted fishes, vegetables and alcoholic drinks for stomach cancer; 3) alcoholic drinks, salted fishes, vegetables, bread, milk, butter, margarine, ketchup, beer and fresh fishes for colon cancer; 4) fresh fishes, salted fishes, salt and sake (2nd class) for rectal cancer; 5) pork, sake (2nd) and green tea for bile ducts cancer; 6) salted fishes, vegetables, alcoholic drinks, oil and fresh fishes for pancreas cancer; 7) beef, chicken, egg and vinegar for liver cancer. (no Refs)

27. NATIONWIDE SURVEY OF MAJOR LIVER DISEASES IN THAILAND ANALYSIS OF 3,305 BIOPSIES AS TO YEAR-END 1978.

Bunyaratvej S, Meenakanit V, Tantachamrun T, Srinawat P, Susilaworn P, Chongchitnan N
Dept. Pathology, Faculty Medicine, Ramathibodi Hosp., Mahidol Univ., Bangkok 4, Thailand
J Med Assoc Thai; 64(9):432-439 1981

Liver cancer, cirrhoses, and inflammatory diseases were surveyed in different regions of Thailand. Liver cancer was more prevalent in the northeast (73.4% of all liver diseases) and in the north (Khonkaen) (42.5%) than in the south (36.8) and central (26.0%) areas. Hepatocellular carcinoma (HPC) was the most common liver cancer in the north (29.5), and cholangiocarcinoma (CC) was most frequently found in the northeast (46.2%). Metastatic carcinoma occurred more in the south (9.4%). HPC affected men 5x more than women, and men also were affected with CC twice as often as women. HPC rate reached a peak in the fifth decade and descended thereafter. Acute viral hepatitis comprised 2.1-4.3% of liver diseases in the different areas, chronic hepatitis 2.1-8.5%. While a high association between CC and opisthorchiasis has been reported, no significant association of viral hepatitis to opisthorchiasis associated CC could be found. Parasitic infections associated with the consumption of raw fish and high levels of nitrates and nitrites in food and surface waterss are discussed as factors in the regional patterns of liver disease. (29 Refs)

GENERAL REVIEWS AND OTHER STUDIES

28. CARCINOGENS OCCURRING NATURALLY IN FOOD.

Hilker DM
Dept. Food Sciences and Human Nutrition, Univ. Hawaii, Henke Hall 224, 1800 East-West Road, Honolulu, HI, 96822
Nutr Cancer; 2(4):217-223 1981

The role of carcinogens which are metabolites of microorganisms infecting plants during harvesting and storage, products of the normal metabolism of plants, products of chemical changes occurring during processing, preservation, or preparation, and which are picked up from the environment through contaminated water or soil is discussed. Some of the most potent carcinogens are fungal metabolites such as aflatoxin B1, 4-ipomeanol, luteoskyrin, methylhydrazine, and ethylcarbamate. The pyrrolizidine alkaloids safrol, oil of calamus, cycasin, thiourea, and tannin also have carcinogenic activity. Polycyclic hydrocarbons, particularly benzo(a)pyrene (BP), are commonly found in food. BP can be found in plant food, seafood, and in meats. Polycyclic hydrocarbons including BP are formed by smoking meat and fish. Epidemiological studies indicate the high incidence of gastric cancer in the areas of the world where smoked fish are common in diets. Vitamin A and certain of its analogues can prevent the carcinogenic activity of BP and some other polycyclic aromatic hydrocarbons. The role of vitamin A appears more important when one considers that the diets of people in areas with high incidence of gastric cancer are low in vitamin A. The interrelated effects of vitamin A-poor diets, consumption of carcinogenic hydrocarbons, and the incidence of cancer need further study. Information is also needed on the amount of carcinogenic chemicals an individual must consume in order to exhibit cancer and on the interrelated effects of other dietary constituents. (59 Refs)

29. CHOICE OF METHODS FOR GATHERING NUTRITION INFORMATION.

Tulinius H
Icelandic Cancer Registry, PO Box 523, Reykjavik, Iceland
Nutr Cancer; 2(4):200-202 1981

Methods for obtaining information on nutrition for epidemiology studies, especially on cancer, are reviewed. Difference in requirements between retrospective and prospective studies and problems posed by their applications are discussed. The methods used for nutrition information are classified into categories. Usage of dietary information require different approaches to data collection. (4 Refs)

30. NUTRITION AND CANCER. INTRODUCTORY REMARKS.

Carroll KK
Univ. Western Ontario, Ontario, Canada
Nutr Cancer; 2(4):197-198 1981

Food consumption data, methods of food analysis, animal models, recommendations regarding cancer at specific sites (colon, breast, stomach, other sites), and data on cancer incidence and mortality have been reviewed. No dietary recommendations to the public have been made. Further experiments are needed to clarify the role of diet in carcinogenesis. (no Refs)

31. NUTRITION AND CANCER. REPORT OF A MEETING.

Siebert G
Abt. f. Experimentelle Zahnheilkunde, Universitatsklinik und Poliklinik fur Zahn-, Mund- und Kieferkrankheiten, 8700 Wurzburg, W. Germany
Z Ernaehrungswiss; 20(4):233-246 1981

Reports presented at a symposium (March 1981; Houston, TX) on 'Molecular Correlation Between Nutrition and Cancer' are summarized. The following general topics were considered at the symposium: formation of carcinogens during the manufacture and preparation of food; inhibition of the ef fects of carcinogens through essential nutrients (vitamins A, C, and E; selenium); dietary fats and cancer; nutrition and metabolism in cancer patients; and nutritional requirements of cancer cells. (no Refs) (In GER)

* U. S. GOVERNMENT PRINTING OFFICE : 1982—361-847/120

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. AN EVALUATION OF THE GENETIC TOXICITY OF EPICHLORHYDRIN. A REPORT OF AN EXPERT GROUP OF THE INTERNATIONAL COMMISSION FOR PROTECTION AGAINST ENVIRONMENTAL MUTAGENS AND CARCINOGENS.
Sram RJ, Tomatis L, Bridges BA, Clemmesen J
(c/o Lohman), Medical Biological Lab. TNO, P.O. Box 45, 2280 AA Rijswijk, The Netherlands
Mutat Res; 87(3):299-319 1981

2. ETHYLENE DIBROMIDE (EDB). (REVISED).
NIOSH Document Development Branch
Robert A. Taft Lab., National Inst. Occupational Safety and Health, 4676 Columbia Parkway, Cincinnati, OH, 45226
Curr Intell Bull NIOSH (37): 13 pp., 1981.

3. CHEMICAL CARCINOGENESIS AND SMOKING.
Farber E
Dept. Pathology, Univ. Toronto, Banting Inst., 100 College St., Toronto, Ontario M5G 1L5, Canada
Can J Public Health; 72(6):381-385 1981

RADIATION CARCINOGENESIS

4. CELLULAR TRANSFORMATION BY RADIATION: INDUCTION, PROMOTION, AND INHIBITION.
Borek C
Radiological Res. Lab., Cancer Center/Inst. Cancer Res., Columbia Univ. Coll. Physicians and Surgeons, New York, NY, 10032
J Supramol Struct Cell Biochem; 16(4):311-336 1981

5. EFFECTS OF IONIZING RADIATION ON THE DEVELOPING EMBRYO AND FETUS: A REVIEW.
Hoffman DA, Felten RP, Cyr WH
Bureau Radiological Health, Rockville, MD
Effects of Ionizing Radiation on the Developing Embryo and Fetus: A Review. Available from National Technical Information Service, Springfield, VA. as PB82-115379., 160 pp., 1981.

DIETARY FACTORS IN CARCINOGENESIS

6. BETA-CARBOLINES, PSYCHOACTIVE COMPOUNDS IN THE MAMMALIAN BODY.
Airaksinen MM, Kari I
Dept. Pharmacology, Univ. Kuopio, P.O. Box 138, SF-70101 Kuopio 10, Finland
Med Biol; 59(4):190-211 1981

7. DIET AND CANCER OF THE LARGE BOWEL.
Zaridze DG
Analytical Epidemiology Programme, Div. Epidemiology and Biostatistics, International Agency for Res. on Cancer, 150 Cours Albert Thomas, 69372 Lyon, Cedex 2, France
Nutr Cancer; 2(4):241-249 1981

8. INFLUENCE OF DIET ON MAMMARY CANCER.
Carroll KK
Dept. Biochemistry, Univ. Western Ontario, London, Ontario, N6A 5C1, Canada
Nutr Cancer; 2(4):232-236 1981

9. ARTIFICIAL SWEETENERS IN RELATION TO THE EPIDEMIOLOGY OF BLADDER CANCER.
Howe GR, Burch JD
NCI Canada Epidemiology Unit, Univ. Toronto, Ontario, Canada
Nutr Cancer; 2(4):213-216 1981

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

10. EPIDEMIOLOGY AND THE STUDY OF CANCER.
Woods NF, Woods JS
Dept. Physiological Nursing, Sch. Nursing, Univ. Washington, Seattle, WA
Cancer Nursing. Marino LB, ed. St. Louis, The C. V. Mosby Company, 635 pp., 1981.

11. EPIDEMIOLOGY OF CUTANEOUS MALIGNANT MELANOMA.
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MMW; 123(50):1923-1926 1981
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12. THE SMOKING-INDUCED EPIDEMIC.
Doll R
Green Coll. Oxford, Oxford, England
Can J Public Health; 72(6):372-381 1981

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13. HEALTH HAZARDS OF ASBESTOS EXPOSURE.
Kaw JL
Industrial Toxicology Res. Centre, Lucknow 226 001, India
J Sci Ind Res; 40(5):342-346 1981

14. OVERVIEW OF FINNISH EPIDEMIOLOGIC STUDIES ON OCCUPATIONAL CANCER.
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Dept. Epidemiology and Biometry, Inst. Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland
Scand J Work Environ Health; 7(Suppl4):133-139 1981

CELLULAR AND MOLECULAR MECHANISMS IN CARCINOGENESIS

15. ALKYLATION OF DNA AND TISSUE SPECIFICITY IN NITROSAMINE CARCINOGENESIS.
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J Supramol Struct Cell Biochem; 17(3):259-273 1981

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

16. TRANSFORMATION INDUCED BY HERPES SIMPLEX VIRUS: A POTENTIALLY NOVEL TYPE OF VIRUS-CELL INTERACTION.
Hampar B
Lab. Molecular Virology, NCI, Frederick, MD, 21701
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17. ONCOGENESIS BY RNA TUMOR VIRUSES.
Koyama T
Dept. Carcinostasis Res., Tokyo Univ. Medical Res. Inst., 4-6-1 Kindai, Minato-ku, Tokyo 108, Japan
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18. POLYOMA T ANTIGENS.
Eckhart W
Tumor Virology Lab., Salk Inst., San Diego, CA, 92138
Adv Cancer Res; 35:1-25 1981

METHODOLOGY IN CARCINOGENESIS RESEARCH

19. EPIDEMIOLOGICAL ASSESSMENT OF DIETARY INTAKE.
James WP, Bingham SA, Cole TJ
Medical Res. Council's Dunn Clinical Nutrition Center, Adenbrooke's Hosp., Cambridge, England
Nutr Cancer; 2(4):203-212 1981

20. ASSESSMENT OF TECHNOLOGIES FOR DETERMINING CANCER RISKS FROM THE ENVIRONMENT.
Office of Technology Assessment
Office of Technology Assessment, Washington, DC
Assessment of Technologies for Determining Cancer Risks from the Environment. Available from National Technical Information Service, Springfield, VA, as PB81-235400:, 243 pp., 1981.

21. ANTIBODIES SPECIFIC FOR DNA COMPONENTS STRUCTURALLY MODIFIED BY CHEMICAL CARCINOGENS.
Muller R, Rajewsky MF
(c/o Rajewsky) Institut fur Zellbiologie (Tumorforschung), Universitat Essen (GH), Hufelandstrasse 55, Essen 1, W. Germany
J Cancer Res Clin Oncol; 102(2):99-113 1981

22. APPLICATION OF THE MICROBIAL MUTATION TEST IN OCCUPATIONAL HEALTH AND ITS FUTURE PROBLEMS.
Takemura N
Dept. Public Health, Jikei Univ., 25-8, Nishi-Shimbashi 2-chome, Minato-ku, Tokyo, 105, Japan
Sangyo Igaku; 23(4):355-365 1981
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23. ARACHIDONIC ACID TRANSFORMATION AND TUMOR PRODUCTION.
Levine L
Dept. Biochemistry, Brandeis Univ., Waltham, MA, 02254
Adv Cancer Res; 35:49-79 1981

24. POLYAMINES IN MAMMALIAN TUMORS PART I.
Scalabrino G, Ferioli ME
Inst. General Pathology, Univ. Milan, 20133 Milan, Italy
Adv Cancer Res; 35:151-268 1981

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ICRDB CANCERGRAM

DIETARY ASPECTS OF CARCINOGENESIS

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STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. MOLD PRODUCING CARCINOGENIC TOXINS IN FOOD PRODUCTS.

Preda N
Institutul de medicina si farmacie, Cluj-Napoca, Romania
Rev Chir [Oncol]; 20(1):1-9 1981

Studies on natural carcinogenic substances, carcinogenic mycotoxins, their occurrence and limit values in food products are reviewed. The known carcinogenic mycotoxins include the

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aflatoxins, patulin, sterigmatocystin, luteoskirin, penicillic acid, and cyclopiazonic acid. The maximum allowable aflatoxin B1 concentrations in food products in different countries are in the range of 0-600 ppb. (14 Refs) (In RUM)

2. AFLATOXINS, B1, B2, G1, G2 IN PRIMARY LIVER CELL CARCINOMA.

Onyemelukwe GC, Ogbadu GH, Salifu A
Dept. Medicine, Ahmadu Bellow Univ., Zaria, Nigeria
Toxicol Lett; 10(2):309-312 1982

The presence or absence of circulating aflatoxins and other mycotoxins in adults (25-65 yr old) with primary liver cell carcinoma (PLCC) was investigated. Serum and urine samples were obtained from each of 20 and 7 PLCC patients, respectively. Serum aflatoxin levels were determined by Velasco Fluorotoxin meter. Mean serum aflatoxin, B1, B2, G1, and G2 levels were 0.069, 0.095, 0.083, and 0.040 ug/ml, respectively. Aflatoxin B1 levels greater than 0.15 ug/ml were measured in three patients; however, higher aflatoxin levels may have been present due to 24 hr serum clearance values of 0.025 to 0.050 ug/ml in these subjects. Hepatitis B surface antigen was detected in eight patients. Two other fungal metabolites, zearalenone, associated with cervical cancer in mice, and Phoma sorghina metabolite, associated with hemorrhagic thrombocytopenia, were identified in two separate urine specimens. Although evidence of aflatoxin carcinogenicity in man is inconclusive, foodstuffs should be protected from excessive aflatoxin contamination. (14 Refs)

3. CONSUMPTION OF POISONOUS PLANTS (SENECIO JACOBAEA, SYMPHYTUM OFFICINALE, PTERIDIUM AQUILINUM, HYPERICUM PERFORATUM) BY RATS: CHRONIC TOXICITY, MINERAL METABOLISM, AND HEPATIC DRUG-METABOLIZING ENZYMES.

Garrett BJ, Cheeke PR, Miranda CL, Goeger DE, Buhler DR
Dept. Animal Science, Oregon State Univ., Corvallis, OR, 97331
Toxicol Lett; 10(2/3):183-188 1982

The effects of the carcinogenic plant comfrey (Symphytum officinale), tansy ragwort (Senecio jacobaea), and bracken (Pteridium aquilinum) on the activity of hepatic drug metabolizing enzymes in rats were investigated. Tansy ragwort and bracken increased the activity of glutathione transferase and epoxide hydrolase. Comfrey and alfalfa increased the activity of aminopyrine N-demethylase. Neither bracken nor St. John's wort (Hypericum perforatum) potentiated the toxicity of tansy ragwort as indicated by rat survival time. Apparently no major toxic interaction exists between tansy ragwort and bracken or St. John's wort. (13 Refs)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

4. ARSENIC-RELATED HEPATIC ANGIOSARCOMA.

Falk H, Caldwell GG, Ishak KG, Thomas LB, Popper H
Chronic Diseases Div., Centers Disease Control, 1600 Clifton Road, NE, Atlanta, GA, 30333
Am J Industr Med; 2(1):43-50 1981

This epidemiological study (highlights only) was conducted to provide additional evidence for arsenic being one cause of hepatic angiosarcoma (HAS). Pathological samples from 350 cases originally diagnosed as HAS in the United States, mostly during 1964-1974, were reexamined. Epidemiological followups (published separately) were made on the 168 confirmed cases involving deaths during 1964-1974. Six case s were attributed to arsenic exposure, five adults from therapy for asthma or psoriasis involving imbibing Fowler's medicine (potassium arsenite), and one child with environmental exposure. Of the five therapy cases (four males and one female, 41-58 yr old), length of therapy and latency period were known only for the males, 10-17 yr and 12-24 yr, respectively. Idiopathic HAS tended to appear much later in life, in the 70s. Four male farmers known to have used Paris green or other unspecified arsenicals for long periods were 68-79 yr old at death from HAS. Two other confirmed cases with deaths prior to 1964 also involved males aged 42 and 50 yr with histories of 6 and less than 15 yr, respectively. The results were taken as supportive of a 1957 report involving autopsies on German vineyard workers, some dying from HAS and all having both used arsenical insecticides and regularly imbibed a beverage made from the grape skins. (27 Refs)

5. AN ENVIRONMENTAL HEALTH SURVEY OF DRINKING WATER CONTAMINATION BY LEACHATE FROM A PESTICIDE WASTE DUMP IN HARDEMAN COUNTY, TENNESSEE.

Clark CS, Meyer CR, Gartside PS, Majeti VA, Specker B, Balistreri WF, Elia VJ
Dept. Environmental Health, Univ. Cincinnati, 3223 Eden Ave., Cincinnati, OH, 45267
Arch Environ Health; 37(1):9-18 1982

Ground water that was used as a source of potable water by residents of a small community became contaminated by leachate from a 300,000 barrel pesticide waste dump. An environmental health survey of the residents and an apparent control group was conducted to determine if any adverse health effects resulting from exposure to the toxic compounds, many of which were hepatotoxins, could be detected. The survey utilized a health questionnaire, a clinical examination, and a biochemical screening. The latter included analysis of serum for liver and kidney function parameters, determination of hepatitis A and B serology, and bile acid determinations on fasting and postprandial serum and urine specimens. Air and water from representative homes and urine from study participants were analyzed for selected organic compounds. The biochemical screening was conducted on two occasions 2 mo apart. The initial hepatic profile testing revealed elevated concentrations of the serum enzymes, alkaline phosphatase and serum glutamic oxaloacetic transaminase, in residents who had used the contaminated water. During follow-up testing of these same persons 2 mo later, these values were significantly reduced, as were postprandial serum concentrations of the bile acid cholyglycine. Six individuals in the exposed group had slight hepatomegaly compared to one individual in the intermediate exposure group and none in the controls. Concentrations of carbon tetrachloride were as high as 18,700 ug/L in the well water samples. The biochemical and clinical observations are suggest ive of a transitory liver injury probably related to exposure to the contaminated drinking water. (Author abstract) (24 Refs)

6. A REVIEW OF TRANSFER FACTORS FOR ASSESSING THE DOSE FROM RADIONUCLIDES IN AGRICULTURAL PRODUCTS.

Ng YC
Environmental Sciences Div., Lawrence Livermore Natl. Lab., P. O. Box 5507, Livermore, CA, 94550
Nucl Saf; 23(1):57-71 1982

The dose to man from radionuclides released to the environment is generally assessed with mathematical models that require transfer factors as input parameters to predict the concentration of radionuclides in foodstuffs. This article summarizes recent attempts to review the worldwide literature and derive updated transfer factors to predict concentrations of radionuclides in terrestrial foods using equilibrium models. Updated transfer coefficients to predict the concentration of a radionuclide in cow's milk and other animal products from that in feed are presented as well as concentration factors to predict the concentration of a nuclide in a food or feed crop from that in soil. Comparing the updated transfer coefficients with those in existing tables leads to suggested changes in the transfer coefficients for milk and beef. Soil-to-plant concentration factors are extremely variable, which limits the usefulness of a single concentration factor to predict the uptake of a radionuclide into crops from soil. The potentially large uncertainty associated with predicting the uptake of radionuclides by plants from soil at a particular location may be reduced by considering the dominant crops and soil types in the area and how various soil properties affect the concentration factor. The updated transfer factors may be useful in assessing transport through terrestrial food chains when site-specific information is not available. In addition, they provide a basis for systematically updating existing tables of transfer factors for generic assessments. (Author abstract) (48 Refs)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

7. ISOLATION AND IDENTIFICATION OF KAHWEOL PALMITATE AND CAFESTOL PALMITATE AS ACTIVE CONSTITUENTS OF GREEN COFFEE BEANS THAT ENHANCE GLUTATHIONE S-TRANSFERASE ACTIVITY IN THE MOUSE.

Lam LK, Sparnins VL, Wattenberg LW
Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN, 55455
Cancer Res; 42(4):1193-1198 1982

Glutathione S-transferase is a major detoxification enzyme system that catalyzes the binding of a variety of electrophiles, including reactive forms of chemical carcinogens, to glutathione (GSH). Green coffee beans fed in the diet induce increased GSH S-transferase activity in the mucosa of the small intestine and in the liver of mice. A potent compound that induces increased GSH S-transferase activity was isolated from green coffee beans and identified as kahweol palmitate. The corresponding free alcohol, kahwe ol, and its synthetic monoacetate are also potent inducers of the activity of GSH S-transferase. A similar diterpene ester, cafestol palmitate, isolated from green coffee beans was active but less so than was kahweol palmitate. Likewise, the corresponding alcohol, cafestol, and its monoacetate showed moderate potency as inducers of increased GSH S-transferase activity. Kahweol palmitate and cafestol palmitate were extracted from green coffee beans into petroleum ether. The petroleum ether extract was fractionated by preparative normal-phase and reverse-phase liquid chromatographies successively. Final purification with silver nitrate-impregnated thin-layer chromatography yielded the pure palmitates of cafestol and kahweol. The structures were determined by examination of the spectroscopic data of the esters and their parent alcohols and by derivative comparison. (Author abstract) (22 Refs)

8. MONITORING N-NITROSAMINO ACIDS EXCRETED IN THE URINE AND FECES OF RATS AS AN INDEX FOR ENDOGENOUS NITROSATION.

Ohshima H, Bereziat JC, Bartsch H
Programme Environmental Carcinogens and Host Factors, International Agency Res. on Cancer, 150, cours Albert-Thomas, F-69372 Lyon Cedex 08, France
Carcinogenesis; 3(1):115-120 1982

A simple and sensitive method for the quantitative estimation of endogenous N-nitrosation in rats has been developed. This approach is based on the findings that N-nitrosamino acids (eg, nitrosoproline (NPRO), nitrosohydroxyproline (NHPRO) and nitrososarcosine (NSAR)) when administered orally to rats, are excreted unchanged almost quantitatively (88-96% of the dose) in the urine and feces. After sequential administration of a nitrosatable amino acid and sodium nitrite the nitrosamino acid excreted in the urine and feces was analyzed. The amount of NPRO excreted in the urine of rats was proportional to the dose of proline and to the square of the nitrite dose. Co-administration of ascorbic acid and alpha-tocopherol together with the precursors decreased the urinary NPRO whereas thiocyanate increased the yield. After feeding an amino acid precursor and nitrite, the yield of nitrosamino acids formed in vivo and excreted in the urine increased in the order: NPRO less than NSAR less than NHPRO. The same order was seen when the nitrosation rates of the amino acids in vitro were compared. Thus N-nitrosation in vivo in rats occurs via a similar mechanism as observed in vitro. Monitoring of N-nitrosamino acids excreted in the urine and feces thus appears to provide a valuable index for endogenous N-nitrosation. (Author abstract) (31 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

9. FAT, FIBER, AND OTHER MODIFIERS OF INTESTINAL CARCINOGENESIS: A STRATEGY FOR PREVENTION.

Nigro ND, Van Soest PJ, Kingston DG, Bruce WR, Tannenbaum SR, Hill MJ
Dept. Surgery, Sch. Medicine, Wayne State Univ., Detroit, MI, 48201
Banbury Rep; 7:83-93 1981

Published reports on the effects of diet on intestinal carcinogenesis in rats (a process requiring a carcinogen) suggest that the influence of dietary fiber is much smaller than the influence of dietary fat. A high-fiber diet shows a protective effect only when the dietary fat level is low. The effect of a high-fat diet may be mediated through the presence of large amounts of steroids in the large intestine, interacting with the bacterial flora. Epidemiologic data on colorectal cancer in populations having different dietary habits tend to support the results of animal experiments. Further studies are needed to define the

relative proportions of dietary fat and fiber for the optimal prevention of colorectal cancer. If only the levels of dietary fat and fiber are changed, it is clear that the changes must be substantial; one report suggests that a 50% reduction in fat and the consumption of bran would be required to reduce the incidence of colorectal cancer in a high-incidence population. Such drastic changes are unlikely to be accepted by the general population or even persons at high risk. It is suggested that a diet containing bran, a less stringently reduced amount of fat, and vegetables inducing benzopyrene hydroxylase in the large intestine (including cabbage, cauliflower, broccoli, and brussels sprouts) may be more acceptable, at least by persons at high risk of colorectal cancer. (28 Refs)

10. IMPLICATIONS OF DIETARY FIBER BREAKDOWN IN THE HUMAN COLON.

Cummings JH, Stephen AM, Branch WJ
MRC Dunn Clinical Nutrition Centre, Old Addenbrookes Hosp., Cambridge, CB2 1QE, England
Banbury Rep; 7:71-81 1981

Metabolism of fiber in the colon and implications for human carcinogenesis are reviewed. Possible hypotheses for the mechanism of the protective effect can be obtained from examining data on the dominant effects of fiber on colorectal function. Dietary fiber is extensively degraded in the colon, leading to production of short-chain fatty acids (butyrate) which may affect colonic cell function. Fiber breakdown also stimulates microbial growth in the colon, leading to increased bulk of the colonic content, faster transit rate, and rerouting of nitrogen metabolism. Fiber fermentation in the colon diverts nitrogen metabolism from ammonia formation (ammonia is more toxic to normal cells than transformed cells) to bacterial protein synthesis. If endogenous nitrite synthesis occurs in the human colon, it may arise from the oxidation of ammonia. The optimum pH for bacterial nitrification in pure cultures and natural ecosystems is alkaline (7.0-8.5). A metabolic consequence of a high-fiber diet may be reducing the colonic pH to a level unfavorable to nitrite synthesis from ammonia, leading to a reduced formation of N-nitroso compounds. The urinary excretion of protein metabolites other than ammonia declines when wheat fiber is added to the diet, showing another change in the direction of nitrogen metabolism in the colon. Any or all these events may be important in protection from colorectal cancer. (56 Refs)

11. RETINOL (VITAMIN A) INHIBITS SISTER CHROMATID EXCHANGES AND CELL CYCLE DELAY INDUCED BY CYCLOPHOSPHAMIDE AND AFLATOXIN B1 IN CHINESE HAMSTER V79 CELLS.

Huang CC, Hsueh JL, Chen HH, Batt TR
Dept. Experimental Biology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263
Carcinogenesis; 3(1):1-5 1982

Retinol (vitamin A alcohol) at the doses tested neither induced an increase of frequency of sister chromatid exchanges (SCE) nor cell cycle delay in Chinese hamster V79 cells with or without the metabolic activation of S-9 mix. However, it inhibited SCE frequencies and cell cycle delay in V79 cells induced by the indirect mutagen cyclophosphamide or aflatoxin B1. The inhibition was found to be dose and time dependent. These results suggest that retinol itself may have no direct effect on the genetic materials but rather exert its effects possibly by inhibiting the metabolic activation of an indirect mutagen or carcinogen. Thus, the antitumor activities of retinoids may not be limited to the widely accepted role of the preventing the promotion step but also the initiation step of carcinogenesis. (Author abstract) (33 Refs)

12. RETINOL (VITAMIN A) AS A MODIFIER OF 2-AMINOFLUORENE AND 2-ACETYL-AMINOFLUORENE MUTAGENESIS IN THE SALMONELLA/MICROSOME ASSAY.

Busk L, Ahlborg UG
Toxicology Lab., Natl. Food Admin., Box 622, S-751 26 Uppsala, Sweden
Arch Toxicol; 49(2):169-174 1982

Vitamin A (retinol) has been demonstrated to modify the mutagenic activity of the aromatic amines, 2-aminofluorene (2AF) and 2-acetylaminofluorene (2AAF) when added to the Ames Salmonella/mammalian microsome assay. Low amounts of retinol (2-20 ug/plate) increased the mutagenicity of both 2AF and 2AAF. At higher doses (50-150 ug/plates) the mutagenicity of 2AAF remained unchanged while the mutagenicity of 2AF gradually decreased. The present data do not support the hypothesis that retinol generally acts as an inhibitor of in vitro metabolic activation of procarcinogens. (Author abstract) (16 Refs)

13. EFFECTS OF 13-CIS RETINOIDS ON PREMALIGNANT AND MALIGNANT CELLS OF LUNG ORIGIN.

Saccomanno G, Moran PG, Schmidt R, Hartshorn DF, Brian DA, Dreher WH, Sowada BJ
Dept. Pathology, St. Mary's Hosp. and Medical Center, P.O. Box 1628, Grand Junction, CO, 81502
Acta Cytol (Baltimore); 26(1):78-85 1982

Deficiency of vitamin A has been shown to cause squamous metaplasia of the epithelium of the tracheobronchial tree. Vitamin A analogues, such as retinoic acid, have been shown to be effective in the treatment of various skin disorders in humans; these compounds have also caused a reduction in the incidence of carcinogen-induced tumors in experimental animals. For these reasons, a clinical trial with orally administered 13-CIS retinoic acid was conducted in 26 patients with varying degrees of cytologic abnormalities in sputum, ranging from moderately atypical metaplasia to overt carcinoma. The drug was administered in daily doses from 1 to 2.5 mg/kg or until toxicity was observed. The cytologic effects of the drug were monitored in sputum. The treatment did not affect the quantitative aspects of sputum cytology. However, degenerative alterations were observed in many of the abnormal cells. Although the experience with 13-CIS retinoic acid reported in this paper is too small for statistical analysis, the cytotoxic effects observed are sufficiently intriguing to suggest further studies in humans and experimental animals. (Author abstract) (25 Refs)

14. A STUDY ON THE ETIOLOGICAL FACTORS OF BILHARZIAL BLADDER CANCER IN EGYPT: 4. BETA-CAROTENE AND VITAMIN A LEVEL IN SERUM.

El-Aaser AA, El-Merzebani MM, Abdel-Reheem KA, Hamza BM
Natl. Cancer Inst., Kasr Aini St., Foum Kaleg, Cairo, Egypt
Tumori; 68(1):19-22 1982

The possible role of vitamin A in the pathogenesis of bilharzial bladder cancer among Egyptians, particularly as it relates to the histopathologic tumor type, was investigated. Bilharzial patients and bladder cancer patients with squamous cell carcinoma, the most prevalent type in Egypt, showed significantly lower levels of vitamin A than normal male subjects. In con-

trast, bladder cancer patients with transitional cell carcinoma had levels that were not significantly different from normal male subjects. The possible role of vitamin A in the etiology of bilharzial bladder cancer is discussed. (Author abstract) (22 Refs)

15. EFFECTS OF DIETARY SELENIUM AND VITAMIN E ON COVALENT BINDING OF AFLATOXIN TO CHICK LIVER CELL MACROMOLECULES.

Chen J, Goetchius MP, Combs GF, Campbell TC
(c/o Campbell) Div. Nutrition Sciences, Cornell Univ., Ithaca, NY, 14853
J Nutr; 112(2):350-355 1982

Day-old single comb white Leghorn chicks of both sexes maternally depleted in selenium (Se) and vitamin E (VE) were fed a low Se and VE-free semipurified basal diet or that diet supplemented with graded levels of Se (0.2-20.0 ppm as Na2SeO3) or VE (100 IU/g as all-rac-alpha-tocopheryl acetate), or both. At 14 days of age, chicks were given 1 mg/kg [3H]aflatoxin B1 (AFB1) ip and killed either 2 or 24 hr later. Covalent binding of AFB1 to liver DNA and RNA in chicks fed the basal diet was significantly greater than in chicks supplemented with Se or VE, or both. Phenobarbital treatment prior to administration of AFB1 decreased adduct formation in most groups, and abolished differences in adduct formation due to diet. These re sults suggest that combined Se-VE deficiency enhances activation or inhibits detoxification of AFB1 in vivo. (Author abstract) (29 Refs)

16. EFFECTS OF DIETARY SELENIUM ON THE METABOLISM AND EXCRETION OF 2-ACETYLAMINOFLUORENE IN THE RAT.

Besbris HJ, Wortzman MS, Cohen AM
Sch. Pharmacy, Univ. Southern California, 1985 Zonal Ave., Los Angeles, CA, 90033
J Toxicol Environ Health; 9(1):63-76 1982

Effects of selenium on metabolism of 2-acetylaminofluorene (AAF) in male weanling CD rats were investigated. Animals were fed either a diet deficient in Se or the same diet supplemented with 0.5 ppm Se as sodium selenite. Hepatic microsomal metabolism of [9-14C]AAF (1 mCi/millimole) was measured in vitro. Microsomes from Se-deficient rats generated significantly less 3-OH-AAF than those from Se-supplemented animals. Only small amounts of N-OH-AAF were produced and no difference was noted between the two groups of animals. Moderately increased activity of glucuronyltransferase occurred in Se-deficient rats while hepatic soluble sulfotransferase activity was the same in both groups. Se-deficient rats excreted more of the given dose of AAF than did Se-supplemented rats; the increase was due to glucuronic acid- and sulfuric acid-conjugated metabolites and non-ether-extractable, water-soluble metabolites. Se-deficient rats excreted more N-OH-AAF, 7-OH-AAF, and 3-OH-AAF, while Se-supplemented rats excreted more 5-OH-AAF. Se-deficient rats excreted more of the administered dose of AAF as 7-OH-AAF than did Se-supplemented animals. A protective effect of dietary Se against AAF-induced hepatocarcinogenesis may be due in part to the inhibition of N-hydroxy-AAF production in vivo. (45 Refs)

17. EFFECT OF COMBINED AND SEPARATE ADMINISTRATION OF EXOGENOUS VITAMINS ON THE INCIDENCE OF CHROMOSOME ABERRATIONS IN RATS SUBJECTED TO SUBACUTE EXPOSURE TO SODIUM FLUORIDE.

Akhundov VIu, Aliev AA, Kul'gavin AE, Sarina TN
Inst. Botany, Baku, USSR
Izv Akad Nauk Azerb [Biol]; (4):3-5 1981

Potential antimutagenic activity of vitamins E, A, and C was studied in random-bred albino rats subjected to po administration of sodium fluoride (SF) at 0.1 LD50 (0.32 mg/100 g). SF increased the incidence of chromosome aberrations in the bone marrow. Daily, po administration of vitamin E alone or in combination with vitamins A and C reduced the mutagenic effect of SF. (2 Refs) (In RUS)

18. INHIBITORS OF GASTROINTESTINAL NEOPLASIA.

Wattenberg LW, Lipkin M, Goldin BR, Tannenbaum SR, Magee PN, Balish E
Dept. Lab. Medicine and Pathology, Univ. Minnesota Medical Sch., Minneapolis, MN, 55455
Banbury Rep; 7:153-165 1981

Chemical carcinogenesis in the mouse forestomach is inhibited by a wide variety of agents, including phenols, lactones, disulfiram (DSM) and derivatives, aromatic isothiocyanates, indoles, a nd sodium cyanate. Intestinal carcinogenesis in rats and mice is inhibited by DSM and related compounds, butylated hydroxyanisole, butylated hydroxytoluene, vitamin E, and selenium salts. Many plant materials consumed by humans contain quercetin, which induces intestinal and bladder tumors in rats and may be the carcinogenic component of bracken fern. The addition of phenothiazine to the diet can inhibit the action of quercetin in mice. The role of intestinal microflora in the production or activation of chemical carcinogens requires further investigation. If the microflora are shown to be involved in intestinal carcinogenesis in humans, antibiotics acting on the intestinal microflora might have a protective effect against intestinal carcinogenesis. Many inhibitors of carcinogenesis have been discovered, often on the basis of empirical work. Some of these inhibitors have the advantage of very low toxicity. (36 Refs)

19. HORMONAL REGULATION OF HUMAN BREAST CANCER CELLS IN VITRO.

Lippman M, Meites J, Welsch CW, Rosen JM, Segaloff A, Koreaman SG, Nandi S, Sirbasku DA
Medical Breast Cancer Section, Medicine Branch, NCI, Bethesda, MD, 20205
Banbury Rep; 8:171-184 1981

Cultured breast cancer cell lines (most of which were derived from neoplastic effusions, often after treatment) differ from the original tumors in several important ways. Experiments with these cell lines must be designed and their results interpreted with great caution. Firm conclusions as to the absence of a given hormone response are particularly difficult to interpret. Positive results are much less ambiguous, but these can also give rise to misinterpretations. Nevertheless, the cell-culture approach provides several advantages in the investigation of hormonal interactions with human breast cancer. In particular, drug- and hormone-resistant variants of wild-type parental cell lines can be used to study the mechanisms of hormone dependence. Hormones now known to affect the growth or differentiated functions of human breast cancer cells (especially the MCF-7 cell line) include estrogens, glucocorticoids,

androgens, thyroid hormones (the ZR-75-1 cell line is absolutely dependent on physiologic levels of triiodothyronine in the medium, and MCF-7 cells are also thyroid hormone dependent), and insulin. Vitamin A derivatives are required for normal epithelial differentiation and inhibit several types of chemical carcinogenesis in rats. Several human breast cancer cell lines contain separable binding activities for retinol and retinoic acid, but clinical trials of 13-cis-retinoic acid in breast cancer patients have not been encouraging. The roles of epidermal growth factor, vitamin D, progesterone, and prolactin in breast cancer growth are not yet clear. (46 Refs)

20. DRUG ANTIMUTAGENESIS (REVIEW).

Zolotareva GN, Akaeva EA

Res. Inst. Biological Testing of Chemical Compounds, Moscow Oblast', USSR

Khim Farm Zh; 15(12):9-14 1981

The results of screening for chemical compounds that reduce the mutagenic properties of drugs but do not affect their therapeutic activity are reviewed. The group of compounds that reduce the level of chemical mutagenesis includes nucleic acids and their metabolites, natural metabolites (spermine, ATP, thiouracil, gibberellic acid), inhibitors of free radical reactions, acridine orange, dimethylsulfoxide, streptomycin, chloramphenicol, actinomycin D, hexamidine, and vitamins A, E. and C. It was emphasized that testing of the potential antimutagenic activity should be carried out in a number of test-systems and in a wide range of concentrations. (92 Refs) (In RUS)

21. CANCER CELL PROCOAGULANT: A NOVEL VITAMIN K-DEPENDENT ACTIVITY.

Delaini F, Colucci M, De Bellis Vitti G, Locati D, Poggi A, Semeraro N, Donati MB

Istituto di Ricerche Farmacologiche 'Mario Negri', Via Eritrea, 62, 20157 Milan, Italy

Thromb Res; 24(3):263-266 1981

Further elaboration of the unexplained, vitamin K-deficiency related, reducing metastasis of 3LL tumors was reported. Four sets of C57Bl/6J mice, the original source of the 3LL tumor, were used and all given 3LL-cell leg implants at day 0: 1. control with neomycin-supplemented diet, 2. as in 1. but diet deficient in vitamin K, and initiated 7 days before day 0, 3. warfarin present in drinking water from day 7, 4. as in 3. but vitamin K3 also in the water from day i4. All animals were sacrificed on day 17 and the primary tumor cells harvested. The tumor cell procoagulant activity (innate ability of 3LL cells to directly activate coagulation factor X) and thrombotest result were both 2. = 3. much less than 1. = 4. Since it had already been suggested that activated factor X might be responsible for fibrin deposition and thus contribute to metastasis, it was now suggested that the antimetastatic role of warfarin be reevaluated. (8 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

22. MODIFICATION OF AGE-RELATED IMMUNE DECLINE IN MICE DIETARILY RESTRICTED FROM OR AFTER MIDADULTHOOD.

Weindruch R, Gottesman SR, Walford RL

Dept. Pathology, Sch. Medicine, Univ. California, Los Angeles, CA, 90024

Proc Natl Acad Sci USA; 79(3):898-902 1982

Although weaning-initiated dietary restriction of rodents is known to increase max survivorship and inhibit spontaneous late-life disease and immunologic aging, restriction begun in adulthood has been much less thoroughly evaluated. In the present studies, male mice of a long-lived F1 hybrid strain were gradually restricted dietarily beginning at 12 mo or older until their body wt stabilized at 60-70% of controls. Underfeeding decreased the number of nucleated cells per spleen but increased th e percentage of T cells. For mice restricted at 12, 17, or 22 mo and tested at various ages thereafter, the [3H]thymidine uptake of spleen cells after phytohemagglutinin stimulation significantly exceeded values for age-matched unrestricted controls. Restriction did not, however, alter either splenocyte responses to concanavalin A or to B-cell mitogens or phytohemagglutinin responses of peripheral lymph node cells. In the splenic plaque-forming cell response to injected sheep erythrocytes, restricted and control mice differed more clearly in response kinetics than in peak levels. The splenic cell-mediated lymphocytotoxic response to alloantigens was comparable in old mice (27-29 mo) restricted since 12 mo of age with that of young (5-to 6-mo) controls and was greater than that of age-matched old controls. Spontaneous tumors were observed less frequently in 19-to 25-mo-old mice restricted at 12 mo of age than in mice restricted at 17 mo or in controls. Our results indicate that appropriate food restriction initiated in adulthood influences immunosenescence and spontaneous tumor incidence in a fashion not unlike its weaning-initiated counterpart. (Author abstract) (31 Refs)

23. DIETARY RESTRICTION IN MICE BEGINNING AT 1 YEAR OF AGE: EFFECT ON LIFE-SPAN AND SPONTANEOUS CANCER INCIDENCE.

Weindruch R, Walford RL

Dept. Pathology, Univ. California Los Angeles, Sch. Medicine, Los Angeles, CA, 90024

Science; 215(4538):1415-1418 1982

Lifelong dietary restriction beginning at 3 to 6 wk of age in rodents is known to decelerate the rate of aging, increase mean and max life-spans, and inhibit the occurrence of many spontaneous cancers. Little is known about the effects of dietary restriction started in middle age. In the experiments now reported the food intake of 12-to 13-mo-old mice of two long-lived strains was restricted by using nutrient-enriched diets in accordance with the concept of 'undernutrition without malnutrition.' The mice on the restricted diet averaged 10 to 20% increases in mean and max survival times compared to the control mice. Spontaneous lymphoma was inhibited by the food restriction. (Author abstract) (26 Refs)

24. INTERACTION OF DIETARY FAT AND THE THYMUS IN THE INDUCTION OF MAMMARY TUMORS BY 7,12-DIMETHYLBENZ(A)ANTHRACENE.

Wagner DA, Naylor PH, Kim U, Shea W, Ip C, Ip MM
(c/o M. Ip) Dept. Experimental Therapeutics, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263
Cancer Res; 42(4):1266-1273 1982

The interaction of dietary fat and the thymus in the induction of mammary tumors by dimethylbenz(a)anthracene has been examined in female Sprague-Dawley rats. In these experiments, rats fed diets of 0.5% (low fat), 5% (normal fat), or 20% (high fat) corn oil from weaning (21 days of age) were thymectomized or sham thymectomized at 35 days of age and were given 5 mg of dimethylbenz(a)anthracene at 55 days of age. Thymectomy exerted a protective effect in rats fed low and normal fat diets, and this was not reversed by Thymosin Fraction V. In high fat-fed rats, tumorigenesis was increased compared to the low fat groups, and in addition, the protective effect of thymectomy was absent. This differential effect of thymectomy could not be explained on the basis of changes in prolactin concentration, since prolactin levels were decreased in all dietary groups. Neither diet nor thymectomy affected corticosterone levels or the estrus cycle of mature rats. Peripheral blood lymphocytes were, however, decreased by both thymectomy and increasing the fat content of the diet. It is hypothesized that the promoting effect of dietary fat on dimethylbenz(a)anthracene-induced mammary tumorigenesis is mediated via the immune system, although a role for the endocrine system still cannot be ruled out. (Author abstract) (66 Refs)

25. FACTORS THAT AFFECT INTESTINAL BACTERIAL ACTIVITY: IMPLICATIONS FOR COLON CARCINOGENESIS.

Goldin BR, Lombardi P, Mayhew J, Gorbach SL, Tannenbaum SR, Wilkins TD, Reddy BS, Goldman P, Hill DH, Montesano R, Magee PN, Strobel HW
Dept. Medicine, Infectious Disease Services, Tufts-New England Medical Center, Boston, MA, 02111
Banbury Rep; 7:41-57 1981

Several experiments in male Fischer rats showed significant dietary effect on fecal beta-glucuronidase (B-Gase), nitroreductase (NRase), and azoreductase (AzRase) activities. Diets containing high levels of beef protein or beef fat were associated with a significant increase in these enzymes. The metabolic conversion of 2-nitronaphthalene (2-NN) or 2-naphthylamine-beta-D-glucuronide to 2-naphthylamine (an index of NRase activity) was significantly higher in beef-fed rats than in grain-fed controls. Dietary supplementation with Lactobacillus acidophilus, tetracycline, or erythromycin reduced fecal enzyme activities in the meat-fed rats to those in controls. The mutagenicity of 2-NN in a NRase-negative Salmonella strain was enhanced in the presence of liver microsomes only by a fecal extract containing NRase activity. Significant dietary effects on fecal enzyme activity were also observed in American adults (20-35 yr old). Lacto-vegetarians and strict vegetarians showed lower fecal B-Gase, NRase, AzRase, and 7-alpha-dehydroxylase activities than omnivores. No major differences were noted between the two vegetarian groups. Dietary supplementation with Lactobacillus caused decreases in fecal B-Gase, NRase, and AzRase activities in omnivores. The enzyme activities returned to baseline values after the supplementation was stopped. The high-animal protein and high-animal fat diets, which are associated with an increased risk of colorectal cancer, caused an increase in bacterial enzymes capable of generating carcinogens in the colon in the rats. The similar association between dietary habits and fecal enzyme activities in humans may provide a partial explanation for the strong positive correlation between daily beef and fat consumption and the incidence of colorectal cancer. (11 Refs)

26. THE METABOLISM OF XENOBIOTICS BY THE INTESTINAL FLORA.

Goldman P
Div. Clinical Pharmacology, Harvard Medical Sch., Beth Israel Hosp., Boston, MA, 02215
Banbury Rep; 7:25-39 1981

In general, the metabolism of xenobiotics involves oxidation and conjugation in the liver, with reducing and deconjugating reactions mediated by the intestinal flora. Several major metabolic reactions have been identified as carried out by the intestinal flora of rodents, but major species differences present a serious obstacle to the use of animal data to clarify the role of the intestinal flora in human colon cancer. The correlation between colon cancer and protein intake is supported by the low reported incidence of colon cancer in vegetarians; but the spouses of colon cancer patients are not at an increased risk of the disease. A susceptibility factor (familial polyposis, ulcerative colitis, etc) may be required before an effect of diet can be discerned. There is experimental evidence that the intestinal flora is essentially fixed for life by contacts during the neonatal period. This suggests that dietary alterations in later life may have little effect on the bacterial composition of an individual's intestinal flora. The differences between the compositions of the fecal flora of residents of countries with different colon cancer incidence rates can be explained in many ways, but they are at least compatible with the concept of a 'national flora' determined by a 'national diet'. It is suggested that studies on possible differences in the metabolism of xenobiotics by persons at different risks of colon cancer might give significant results if the tests involved assays of fecal mutagens rather than the microbial compositions of the intestinal flora. (50 Refs)

27. SOME FACTORS INFLUENCING THE ECOLOGY OF GUT FERMENTATION IN MAN.

Van Soest PJ, Hill MJ, Wattenberg LW
Div. Nutritional Sciences and Dept. Animal Science, Cornell Univ., Ithaca, NY, 14853
Banbury Rep; 7:61-69 1981

In an 80-day experiment, 24 young men were fed a low-fiber basal diet (meat, eggs, mashed potatoes, and white bread) supplemented by high-fiber bread made with coarse of fine bran, cabbage powder, or wood cellulose (max fiber level, 40 g/day). The effects of the dietary fiber on digestive function varied from one fiber type to another. Particle size, hydration capacity, and fermentability were more important than the actual chemical constitution of the fiber source. A significant negative correlation was noted between the intestinal passage rate and fiber digestion. The efficiency of metabolic conversion from fermentabl e fiber to fecal nitrogen was greatest with coarse bran, followed by cabbage, fine bran, and cellulose. The results suggested that metabolism by colonic microorganisms and their net systems might create a metabolic sink, in which potentially toxic substances are incorporated into the microbial cellular mass. Increasing the size of the sink could become a means of detoxification by colonic microflora, since their non-carbohydrate requirements for cellular growth must be scavenged from the intestinal environment. Nitrogenous substances bound in the microbial cells are excreted in the metabolic fecal organic matter. The role of the colonic microflora as related to

the intestinal environment and health may vary according to intestinal ecology and substrate availability. The nitrogen requirement of the intestinal microflora may vary from an ammonia- and amine-producing fermentation (oversupply of nitrogen) to a nitrogen-utilizing situation (insufficient supply) in which urea, ammonia, and perhaps other hazardous substances are utilized in the microbial cell synthesis. (13 Refs)

28. COFFEE, TEA AND CANCER: HOW TO TEST HYPOTHESES ON LIFESTYLE AND DISEASE?.

Olsson H, Brandt L
Onkologiska kliniken, Lund, Sweden
Lakartidningen; 79(4):196-197 1982

Coffee and tea consumption of 550 tumor patients was analyzed based on questionnaire data. The patients included 19 with seminomas, 36 with cancer of the upper part of the digestive tract (lips, oral cavity, epipharynx, hypopharynx and esophagus), 59 with Hodgkin's disease, 64 with tumors of the larynx and lungs, 36 with thyroid tumors, 120 with cancer of the urinary bladder or kidneys, 28 with skin cancer, 94 with breast cancer, 16 with testicular teratomas, 28 with melanomas, 20 with cancer of the lower parts of the digestive tract (stomach, bile ducts, pancreas, colon and rectum), and 18 with tumors of the paranasal sinuses, auditory meatus and parotid glands. The median coffee consumption of the patients was 3-5 cups/day, ie, approx the same as in the general population. Fifty-three per cent of the patients with seminomas, 50% of those with cancer of the upper part of the digestive tract and 47% of those with Hodgkin's disease consumed large amounts of coffee (5 cups/day or more). The median tea consumption of the patients was 0 cup/day. Forty-four per cent of the patients with testicular teratomas, 31% of those with thyroid tumors, 32% of those with seminomas, and 29% of those with Hodgkin's disease consumed large amounts of tea (2 cups/day or more). The findings indicate that hypotheses based on complex data on lifestyle (education, occupational status, living conditions, dietary and smoking habits, and alcohol consumption) should be tested in epidemiological studies, rather than the possible relationship between tea and coffee consumption, on one hand, and certain diseases, on the other hand. (6 Refs) (In SWE)

29. BREAST CANCER AND ALCOHOLIC-BEVERAGE CONSUMPTION.

Rosenberg L, Shapiro S, Slone D, Kaufman DW, Helmrich SP, Miettinen OS, Stolley PD, Levy M, Rosenshein NB, Schottenfeld D, Engle RL
Drug Epidemiology Unit, Boston Univ. Sch. Medicine, 777 Concord Ave., Cambridge, MA, 02138
Lancet; 1(8266):267-271 1982

The relation between breast cancer and alcoholic-beverage consumption was evaluated in a cose-control study of 1,152 women with breast cancer and two groups of control women - 519 with endometrial or ovarian cancer, and 2,702 with nonmalignant disorders. The relative-risk estimate of breast cancer, with allowance for all potential distorting factors, for women who had ever drunk alcoholic beverages relative to those who had never drunk was 1.4 (95% confidence interval, 1.0-2.0) when the comparison group was the group with endometrial or ovarian cancer and 1.9 (1.5-2.4) when the controls who had non-malignant disorders were the comparison group. The association was evident for beer, wine, and spirits. The association was not explained by any of the major known risk factors for breast cancer, but we had no information on dietary factors. The findings support the hypothesis that alcohol consumption, or related dietary factors, increases the risk of breast cancer. (Author abstract) (20 Refs)

30. INTAKE, FORMATION, AND RELEASE OF MUTAGENS BY MAN.

Stich HF, Rosin MP, San RH, Wu CH, Powrie WD, Van Soest PJ, Wattenberg LW, Watson JD, Tannenbaum SR, Wilkins TD, Magee PN, Bruce WR
Environmental Carcinogenesis Unit, British Columbia Cancer Res. Centre, Vancouver, British Columbia V5Z 1L3, Canada
Banbury Rep; 7:247-266 1981

The incidence of stomach cancer was high in Japan during the Edo period of its history, when foodstuffs were not contaminated by industrial or agricultural chemicals. During the past several decades, wher. the uses of these chemicals increased in Japan and elsewhere, the incidences of cancer of the stomach, esophagus, and mouth in Japan declined steadily. Many classes of compounds occurring in fresh, cooked, and stored foodstuffs have been identified as mutagens. The use of food additives, pesticides, and herbicides will undoubtedly add to the mutagenic load of foodstuffs. The available evidence raises several questions: whether foodstuffs comprise the major source of mutagens (and, by implication, carcinogens) in the human body; why the cancer frequency is so low, despite this daily intake of mutagens; and why DNA repair-deficient patients (eg, those with xeroderma pigmentosum) develop skin cancers at an early age but lack a comparable frequency of gastrointestinal cancers. It is difficult to discard the idea that people always ingest enough carcinogens to produce neoplastic cells, but that the occurrence of this event is enhanced or s uppressed by genetic factors, whose role may be greater than hitherto assumed. If mutational events are involved at all in tumor initiation, they may not be the types of events detected by the in vitro bioassays now used. The number of different mutagens in food products, digested foods, and feces is evidently very large. The genetic damage in somatic cells may be the product of the actions of many mutagens rather than of one predominating substance. (31 Refs)

31. AFLATOXIN LEVELS IN SERA OF HEALTHY FIRST TIME RURAL BLOOD DONORS: PRELIMINARY REPORT.

Onyemelukwe GC, Ogbadu G
Dept. Medicine, Ahmadu Bello Univ. Hosp., Zaria, Nigeria
Trans R Soc Trop Med Hyg; 75(6):780-782 1981

Serum levels of aflatoxin B1 (AB1), aflatoxin B2 (AB2), aflatoxin G1 (AG1), and aflatoxin G2 (AG2) were assayed in 20 first-time blood donors (25-40 yr old) and 15 sick persons (controls). The blood donors were all farmers from rural villages of the Guinea Savannah of Nigeria, cultivating and living on local staple foods (millet, sorghum, rice, and yams). The controls (including a man with nasopharyngeal carcinoma and a woman with hydatidiform mole) lived in villages near the hospital. Mean serum aflatoxin (AFL) levels in the blood donors were 0.123, 0.118, and 0.118 ug/ml for AB1, AB2, and AG1, respectively, and 0.064 ug/ml for AG2. Four blood donors showed AB1 levels of 0.2 ug/ml. Hepatitis B surface antigen (HBS-Ag) was present in the serum of 1/20 blood donors, who showed a low serum AFL level and no clinical disease. In the control group, serum AFL levels were significantly lower than in the farmers. The highest AFL levels (0.14 ug/ml of AB1, 0.24 ug/ml of AB2) were observed in the patient with nasopharyngeal cancer. Three patients with liver cirrhosis were positive for HBS-Ag and showed low serum AFL levels. Since liver function tests were normal in the farmers and in the absence of

histological examination of their livers, it was difficult to speculate whether any of them had entered the multistage process of hepatic carcinogenesis. Further research is needed to evaluate the interactions (if any) between AFL and Epstein-Barr, hepatitis B, and cytomegaloviruses. (16 Refs)

32. THE MEANING OF CANCER 'DIETS'.

Jungi WF
Abt. f. Onkologie-Hamatologie, Medizinische Klinik C, CH-9007 St. Gallen, Switzerland
MMW; 124(2):26-28 1982

The rationale and content of diets that are promoted as preventive or therapeutic cancer treatments are discussed. The basis of cancer diets is reviewed with respect to theories concerning differences between normal and malignant cells. The basic principles of several cancer diets are outlined. One metabolically active, cancer-fighting' diet plan calls for eating several small meals, increasing the intake of unrefined carbohydrates and vegetable fiber, increasing the amount of protein from plant s ources, decreasing the consumption of animal fats, and limiting salt intake. Other diets that offer specific suggestions concerning appropriate choices of fat and protein sources, fruits, and vegetables are described. An extreme form of a cancer diet, the healing fast, is also considered. It is noted that, although there is no evidence to prove that these cancer diets can prevent or cure cancer, the relationship of nutritional status to the development and disease course of many forms of cancer has been definitely established. In light of this, and in consideration of the fact that many of these cancer diets contain nutritionally good suggestions, further research on the effects of adjuvant diet therapy is recommended. (21 Refs) (In GER)

33. EPIDEMIOLOGIC TESTS OF HYPOTHESES RELATING DIET TO CANCER.

Graham S, Correa P, Hill MJ, Bruce WR, Wattenberg LW, Stemmermann GN, Reddy BS
Dept. Sociology, State Univ. New York at Buffalo, Buffalo, NY, 14261
Banbury Rep; 7:395-408 1981

Problems with epidemiological studies relating diet to cancer are discussed. People generally cannot remember with accuracy what they ate at a given time in their lives. Interview studies of the subject with and without the spouse, validity studies, and prospective studies are described. Hypotheses relating diet and cancer are reviewed for dietary fiber, vitamin C, Cruciferae, vegetables, fats, coffee, and saccharin. (36 Refs)

34. A LARGE-SCALE COHORT STUDY ON THE RELATIONSHIP BETWEEN DIET AND SELECTED CANCERS OF DIGESTIVE ORGANS.

Hirayama T, Tannenbaum SR, Reddy BS, Goldin BR, Wogan GN, Magee PN, Keefer LK, Correa P
Epidemiology Div., Natl. Cancer Center Res. Inst., Tokyo, Japan
Banbury Rep; 7:409-429 1981

Of 265,118 adults, aged 40 and above, who were followed for 13 yr, 39,127 died. The leading cause of death was cerebral vascular disease followed by cancer. Standardized mortality rates were calculated for each cause of death according to lifestyle variables that had been previously recorded when the subjects were healthy. The relationship between yellow and green vegetable, meat, and rice intake, alcohol consumption, and cigarette smoking, and the risk of certain cancers of the digestive organs was evaluated. (23 Refs)

35. LIVER-FLUKE INFECTION AS AN AETIOLOGICAL FACTOR IN BILE-DUCT CARCINOMA OF MAN.

Flavell DJ
Winches Farm Field Station, London Sch. Hygiene and Tropical Medicine, 395 Hatfield Rd., St. Albans, Herts AL4 0XQ, England
Trans R Soc Trop Med Hyg; 75(6):814-824 1981

Evidence for an etiologic relationship between liver fluke infections (Clonorchis sinensis in southern China, Korea, and Japan; Opisthorchis viverrini in northeast Thailand) and primary cholangiocarcinoma of the liver is discussed. The etiology of cholangiocarcinomas arising in association with th ese trematodes is multifactorial. In the case of Opisthorchis, when freshwater ciprinoid fish containing metacercariae are eaten undercooked or raw, the metacercariae excyst in the duodenum and the juvenile flukes migrate through the common bile duct and extrahepatic biliary system to the intrahepatic bile ducts, where they mature and lay their eggs. For both parasites, the malignant transformation of infected bile ducts may initially involve a high turnover of hyperplastic biliary epithelial cells, caused directly by the parasite. This process may be further stimulated by a low protein intake by the host. The hyperplastic and proliferating bile ducts may be susceptible to the action of carcinogen(s) present at levels too low to induce cholangiocarcinomas in uninfected persons. Such carcinogens may be naturally occurring or they may form in vivo from ingested precursor molecules or endogenous substances. The host's immunologic status may also be affected by the infection, which may play a role in tumorigenesis. (77 Refs)

36. BREAST CANCER: DIET AND HORMONE METABOLISM.

Hill P, Garbaczewski L, Kasumi F, Kuno K, Helman P, Wynder EL, Topp W, Pike MC, Henderson BE, Segaloff A, Sirbasku DA, Korenman SG, Cole P, Zumoff B
Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY, 10595
Banbury Rep; 8:257-277 1981

The effect of diet modification on hormonal metabolism of women at low risk (black South African and Japanese) and women at high risk (North American caucasian) for breast cancer was investigated. Differences in the hormonal profile during the menstrual cycle between the three races of women were noted. Differences in pituitary response in circulating hormone levels and in duration of menstrual cycle occurred when a Western diet was interchanged for a vegetarian diet, and vice versa. The therapeutic value of this observation is unknown. (94 Refs)

GENERAL REVIEWS AND OTHER STUDIES

37. EPIDEMIOLOGY OF THE MOST FREQUENTLY OCCURRING TUMORS OF THE DIGESTIVE SYSTEM.

Naujoks-Heiarich S, Dolle W
Abteilung Innere Medizin I, Universitatsklinik, Otfried-Muller-Strasse 10, D-7400 Tubingen, W. Germany
Internist (Berlin); 23(1):1-9 1982

The epidemiology of esophageal, gastric, and colorectal cancer is reviewed. These types of cancer are described with respect to incidence, variations in incidence between countries, age and sex distribution of patients, lethality, exogenous factors that increase the risk of cancer development, benign conditions

associated with each, and genetic predisposition to cancer development. Exogenous factors that increase the risk of development of esophageal cancer include alcohol and tobacco use; among the benign conditions frequently associated with this type of cancer are achalasia, scleroderma, Plummer-Vinson syndrome, and benign esophageal stricture. Genetic aspects of gastric and colorectal cancer are examined with respect to studies of the incidence of these diseases in Japanese people living in Japan and emigrants of Japanese ancestry. Exogenous factors that increase the risk of developing gastric cancer include occupational exposure to coal dust and exposure to trace elements such as asbestos in drinking water. Pernicious anemia, gastric polyps, and ulcera ventriculi are among the benign conditions associated with this type of cancer. The role of diet in the development of colorectal cancer is emphasized; the relative significance of animal fats, vegetables, and fiber in the diet is considered. The general field of epidemiology is discussed with regard to methodologic problems, including variations between countries in diagnostic methods and criteria as well as problems in documentation. (52 Refs) (In GER)

38. CANCER AND NUTRITION.

Betzler M
Chirurgische Univ.-Klinik, Heidelberg, W. Germany
MMW; 124(2):21 1982

Selected aspects of the relationship between nutrition and cancer in humans are considered. Among the topics discussed are the role of diet in the etiology of certain types of cancer (eg, colon cancer), the clinical observation that many cancer patients are malnourished, the effect of nutritional status on the outcome of treatment, types of nutritional therapy for reversing cancer-related malnutrition, and the possibility that diet alone may have a prophylactic or therapeutic benefit in cancer development. (3 Refs) (In GER)

39. EFFECT OF GEROPROTECTORS ON CARCINOGENESIS.

Anisimov VN
N. N. Petrov Res. Inst. Oncology, Leningrad, USSR
Usp Sovrem Biol; 92(3):455-472 1981

Relationships between the aging and neoplastic transformation are reviewed with special emphasis on the effects of geroprotectors (agents which increase the lifespan) on the incidence and latent period of experimental tumors. On the basis of the mechanism of activity, the geroprotectors can be divided into two groups. Group 1 includes antioxidative agents that prevent the damages leading to a neoplastic cell transformation, and Group 2 includes the agents that inhibit the realization of genetic program of the aging and development of the age-related pathology (antidiabetic biguanides, epithalamine, and low-calorie diet). It was suggested that antioxidative agents do not inhibit the aging per se but rather reduce the effects of environmental factors. The geroprotectors from Group 2 decrease the sensitivity threshold of the hypothalamus to homeostatic signals. (156 Refs) (In RUS)

40. THE TOXICOLOGY OF COCOA AND METHYLXANTHINES: A REVIEW OF THE LITERATURE.

Tarka SM
Life Sciences, Hershey Foods Corp., Corporate Technical Center, Hershey, PA
CRC Crit Rev Toxicol; 9(4):275-312 1982

Toxicology of cocoa and methylxanthines (caffeine, theobromine, and theophylline) was discussed with respect to the following: physical properties; toxic effects of cocoa and theobromine in the diets of various animals; biological effectiveness of the methylxanthines in cocoa; toxicity of methylxanthines; distribution, metabolism, and excretion in animals and humans (metabolism during pregnancy, lactation, and growth; diet and drug influence on methylxanthine metabolism); pharmacological properties of the methylxanthines; effects of methylxanthines on growth and other physiological processes; effects on reproduction; teratogenesis; mutagenesis; chronic toxicity/carcinogenesis; SCOGS report; and fibrocystic breast disease. Research on teratogenic and mutagenic effects of caffeine was thought to be relatively abundant, but there are only a few reports on the carcinogenic effects of caffeine and none on such effects of cocoa powder, theobromine, theophylline, or tea. (203 Refs)

41. BILE SALTS AND OTHER CONSTITUENTS OF THE COLON AS TUMOR PROMOTERS.

Reddy BS, Tannenbaum SR, Montesano R, Correa P, Haenszel W, Wattenberg LW
Div. Nutrition, American Health Foundation, Naylor Dana Inst. for Disease Prevention, Valhalla, NY, 10595
Banbury Rep; 7:345-363 1981

The mechanisms by which dietary components increase the risk of developing colon cancer are reviewed and etiologic factors in large-bowel carcinogenesis are discussed. Dietary fat and fiber affect fecal bile acid excretion, which may then affect carcinogenesis. Cholesterol and its metabolites are mutagenic and have colon-tumor-promoting activity. Fecal mutagens and comutagens in high- and low-risk populations for colon cancer were studied. (63 Refs)

42. BILE ACIDS AND CARCINOMA OF THE LARGE BOWEL.

Raedsch R
Innere Medizin IV, Medizinische Universitatsklinik, Bergheimer Strasse 58, 6900 Heidelberg, W. Germany
Inn Med; 9(1):8-15 1982

Possible correlations between high-fat diets, increased concentration of bile acids in the intestines, and the development of cancer of the colon are discussed. The following aspects of these correlations are considered: metabolism of bile acids, animal models for a relationship between bile acids and colon cancer, bile acids and intestinal bacteria, carcinogenic, cocarcinogenic, and procarcinogenic activities of bile acids, clinical significance of bile acids, and relationship between bile acids and dietary fiber. (74 Refs) (In GER)

43. GASTROINTESTINAL CANCER: ENDOGENOUS FACTORS.

Bruce WR, Correa P, Lipkin M, Tannenbaum SR, Wilkins TD
Ontario Cancer Inst., Toronto, M4X 1K9, Canada
Banbury Rep 7: 468 pp., 1981.

The role of diet in cancer was examined. Sessions on gastrointestinal microbiology (microbiological considerations in experimental studies, effect of diet on human fecal flora, met abolism of xenobiotics by intestinal flora, and intestinal bacterial activity and carcinogenesis), fiber (ecology of gut fermentation in man, dietary fiber breakdown in the human colon role of fat, fiber, and other modifiers of intestinal carcinogenesis in cancer prevention), host response to carcinogens (risk factor profiles for individuals at increased risk for colon cancer, fecal mutagens and colorectal polyps, enzymes of normal, premalignant,

and malignant colonic cells, role of cytochrome P-450 in colon response xenobiotics, inhibitors of gastrointestinal neoplasia, colonic activation of carcinogens), mutagens (in extracts of gastrointestinal mucosa, nitrosation in stomach mucosal tissue of humans and dogs, isolation of mutagens produced in the human colon by bacterial action, structural studies and properties of mutagens isolated from human feces, bacterial fluctuation tests of mutagenic activity in human feces, intake, formation, and release of mutagens by man), N-nitroso compounds (endogenous formation of N-nitroso compounds, analysis of human biological specimens for nitrosamines, block nitrosamine formation using ascorbic acid and alpha-tocopherol, distribution and metabolism of nitrate and nitrite in rats, analysis of nitrate, nitrite, and nitrosamines in human feces, metabolism of nitrate), bile acids and other lipids (bile salts as tumor promoters, bile acids in colorectal carcinogenesis, and analysis of feces for benzopyrene after consumption of charcoal-broiled beef) and epidemiology and design of future studies (epidemiologic tests of hypothesis relating diet and cancer, large-scale cohort study of diet and selected digestive system cancers, and possible application of mutagenesis testing to cancer epidemiology). (715 Refs)

* U. S. GOVERNMENT PRINTING OFFICE : 1982—361-847/122

44. MICROBIOLOGICAL CONSIDERATIONS IN INTERPRETATION OF DATA OBTAINED WITH EXPERIMENTAL ANIMALS.

Wilkins TD
Dept. Anaerobic Microbiology, Virginia Polytechnic Inst. and State Univ., Blacksburg, VA, 24061
Banbury Rep; 7:3-9 1981

The intestinal flora of rodents differs greatly from that of humans. The rodent forestomach is lined with lactobacilli, which may interact with any drug given po. Metabolically active yeasts line the secreting portion of the stomach. Large numbers of fecal bacteria may also be present, despite efforts to prevent coprophagy. The upper small intestine harbors an unusual filamentous bacterium which is poorly understood. These bacteria become attached to the host cell membranes. The first segment of the filament is modified into a specialized attachment cell that actually inserts into the host cell membrane to form intracellular bodies which are released and begin to multiply after they attach to new host cells. The terminal ileum harbors a large population of microorganisms, not closely associated with the mucosa, which metabolize oxidized compounds. Laboratory rats and mice have a small cecum because a laboratory diet provides enough nutrients whereas the cecum of wild rodents is large and harbors microorganisms that ferment complex polysaccharides of plant origin. The cecal and colonic flora of rodents consist largely of large fusiform organisms (mainly clostridia), which are not found in the human colon. Most of these species have not yet been described or named. Major dietary changes have little influence on the intestinal flora of humans, whereas dietary manipulation or starvation can cause drastic changes in the intestinal flora of rats and mice. Investigators studying dietary effects on colonic carcinogenesis in rodents should bear these differences in mind. (16 Refs)

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. IARC MONOGRAPHS ON THE EVALUATION OF THE CARCINOGENIC RISK OF CHEMICALS TO MAN. SOME ANTINEOPLASTIC AND IMMUNOSUPPRESSIVE AGENTS.
Schmahl D
International Agency for Cancer Research, Lyon, France
IARC Mongr Eval Carcinog Risk Chem Hum 26: 411 pp., 1981.

2. BIOCHEMICAL TOXICOLOGY OF BENZENE.
Snyder R, Longacre SL, Witmer CM, Kocsis JJ, Andrews LS, Lee EW
Dept. Pharmacology, Thomas Jefferson Univ., Philadelphia, PA, 19107
Rev Biochem Toxicol; 3:123-153 1981

3. A REVIEW OF THE MUTAGENICITY OF CHROMIUM.
Hatherill JR
Toxicology, Sch. Public Health, Univ. Michigan, Ann Arbor, MI, 48109
Drug Chem Toxicol; 4(3):185-195 1981

4. HORMONES AND BREAST CANCER.
Pike MC, Siiteri PK, Welsch CW
Dept. Family and Preventive Medicine, Univ. Southern California Sch. Medicine, Los Angeles, CA, 90033
Banbury Rep 8: 491 pp., 1981.

5. ORAL CONTRACEPTIVES: VOLUME 5.
Briggs MH, Briggs M
Deakin Univ., Geelong 3217, Australia
Oral Contraceptives: Volume 5. St. Albans, VT, Eden Medical Research Inc., Annual Research Reviews--Endocrinology, 331 pp., 1981.

6. EPIDEMIOLOGICAL STUDIES OF THE ROLE OF EXOGENOUS ESTROGENS IN THE ETIOLOGY OF BREAST CANCER.
Kelsey JL, Bulbrook RD, Korenman SG, Siiteri PK, Henderson BE
Dept. Epidemiology and Public Health, Yale Univ. Sch. Medicine, New Haven, CT, [illegible]10
Banbury Rep; 8:215-228 1981

7. EFFECTS OF LONG-TERM EXPOSURE TO LOW LEVELS OF OZONE: A REVIEW.
Melton CE
Civil Aeromedical Inst., Federal Aviation Admin., Oklahoma City, OK, 73125
Aviat Space Environ Med; 53(2):105-111 1982

8. METABOLIC INTERACTIONS OF HYDROCARBONS WITH MAMMALIAN LUNG.
Smith BR, Bend JR
Lab. Pharmacology, Natl. Inst. Environmental Health Sciences, NIH, Research Triangle Park, NC, 27709
Rev Biochem Toxicol; 3:77-122 1981

9. REVIEW OF EXPERIMENTAL CARCINOGENESIS BY COMPOUNDS RELATED TO VINYL CHLORIDE.
Chu KC, Milman HA
NCI, Bethesda, MD, 20205
Environ Health Perspect; 41:211-220 1981

10. REVIEWS IN BIOCHEMICAL TOXICOLOGY.
Hodgson E, Bend JR, Philpot RM
North Carolina State Univ., Raleigh, NC
Rev Biochem Toxicol 3: 368 pp., 1981.

DIETARY FACTORS IN CARCINOGENESIS

11. ALCOHOL AND CANCER.
MacSween RN
Univ. Dept. Pathology, Western Infirmary, Glasgow, Scotland
Br Med Bull; 38(1):31-33 1982

12. THE TOXICOLOGY OF COCOA AND METHYLXANTHINES: A REVIEW OF THE LITERATURE.
Tarka SM
Life Sciences, Hershey Foods Corp., Corporate Technical Center, Hershey, PA
CRC Crit Rev Toxicol; 9(4):275-312 1982

13. IMPLICATIONS OF DIETARY FIBER BREAKDOWN IN THE HUMAN COLON.
Cummings JH, Stephen AM, Branch WJ
MRC Dunn Clinical Nutrition Centre, Old Addenbrookes Hosp., Cambridge, CB2 1QE, England
Banbury Rep; 7:71-81 1981

14. BILE SALTS AND OTHER CONSTITUENTS OF THE COLON AS TUMOR PROMOTERS.
Reddy BS, Tannenbaum SR, Montesano R, Correa P, Haenszel W, Wattenberg LW
Div. Nutrition, American Health Foundation, Naylor Dana Inst. for Disease Prevention, Valhalla, NY, 10595
Banbury Rep; 7:345-363 1981

15. MOLD PRODUCING CARCINOGENIC TOXINS IN FOOD PRODUCTS.
Preda N
Institutul de medicina si farmacie, Cluj-Napoca, Romania
Rev Chir [Oncol]; 20(1):1-9 1981
In RUM

16. THE MEANING OF CANCER 'DIETS'.
Jungi WF
Abt. f. Onkologie-Hamatologie, Medizinische Klinik C, CH-9007 St. Gallen, Switzerland
MMW; 124(2):26-28 1982
In GER

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

17. RISK FACTORS FOR CERVICAL CANCER.
Hulka BS
Dept. Epidemiology, Rosenau Hall 201H, Univ. North Carolina, Chapel Hill, North Carolina, NC, 27514
J Chronic Dis; 35(1):3-11 1982

18. EPIDEMIOLOGY OF THE MOST FREQUENTLY OCCURRING TUMORS OF THE DIGESTIVE SYSTEM.
Naujoks-Heinrich S, Dolle W
Abteilung Innere Medizin I, Universitatsklinik, Otfried-Muller-Strasse 10, D-7400 Tubingen, W. Germany
Internist (Berlin); 23(1):1-9 1982
In GER

19. BLADDER CANCER.
Skrabanek P, Walsh A
Endocrine Oncology Unit, Mater Misericordiae Hosp., Dublin, Ireland
UICC Tech Rep Ser 60: 192 pp., 1981.

20. RECENT ADVANCES IN CANCER CYTOGENETICS.
Pogosiants EE
All-Union Cancer Res. Center, Moscow, USSR
Genetika; 17(12):2087-2099 1981
In RUS

21. CURRENT STATUS OF CYTOGENETIC STUDIES IN ANIMAL TUMORS WITH SPECIAL REFERENCE TO NONRANDOM CHROMOSOME CHANGES.
Sasaki M
Chromosome Res. Unit, Hokkaido Univ., Faculty Science, Sapporo 060, Japan
Cancer Genet Cytogenet; 5(2):153-172 1982

ENVIRONMENTAL AND OCCUPATIONAL FACTORS IN CARCINOGENESIS

22. ASBESTOS-RELATED DISEASES.
Casey KR, Rom WN, Moatamed F
Building 512, RMCOEH, Univ. Utah Medical Center, Salt Lake City, UT, 84112
Clin Chest Med; 2(2):179-202 1981

23. NONASBESTOS FIBROUS MINERALS.
Lockey JE
Building 512, RMCOEH, Univ. Utah Medical Center, Salt Lake City, UT, 84112
Clin Chest Med; 2(2):203-218 1981

24. REVIEW OF EPIDEMIOLOGIC STUDY RESULTS OF VINYL CHLORIDE-RELATED COMPOUNDS.
Apfeldorf R, Infante PF
Office Carcinogen Identification and Classification, Health Standards, Programs, Occupational Safety and Health Admin. U. S. Dept. Labor, Washington, DC, 20210
Environ Health Perspect; 41:221-226 1981

25. CONCEPTS IN CAUSALITY: CHEMICALLY INDUCED HUMAN URINARY BLADDER CANCER.
Lower GM
Center Human Systems, Inst. Environmental Studies, Univ. Wisconsin, WAKF Building, 610 Walnut St., Madison, WI, 53706
Cancer; 49(5):1056-1066 1982

CELLULAR AND MOLECULAR MECHANISMS IN CARCINOGENESIS

26. MOLECULAR MECHANISM OF ADAPTATION OF CELLS TO LOW LEVELS OF CARCINOGENIC AGENTS.
Villaui G
Laboratoire de Pharmacologie et de Toxicologie Fondamentales, CNRS, 205, route de Narbonne, 31078 Toulouse Cedex, France
Pathol Biol (Paris); 30(2):117-123 1982
In FRE

27. MIGHT CANCER BE A FAILED RESPONSE TO RENEGADE MITOCHONDRIA?
Hartung J
Dept. Anesthesiology, State Univ. New York, Downstate Medical Center, Brooklyn, NY, 11203
J Theor Biol; 94(1):173-178 1982

MODIFICATION OF CARCINOGENESIS

28. EFFECT OF GEROPROTECTORS ON CARCINOGENESIS.
Anisimov VN
N. N. Petrov Res. Inst. Oncology, Leningrad, USSR
Usp Sovrem Biol; 92(3):455-472 1981
In RUS

29. RELATION OF NEUROLEPTIC DRUGS TO DEVELOPMENT AND GROWTH OF MAMMARY TUMORS.
Meites J, Aylsworth CF
Dept. Physiology, Neuroendocrine Res. Lab., Michigan State Univ., East Lansing, MI, 48824
Banbury Rep; 8:365-376 1981

30. THE ACTIONS OF INSULIN AS A HORMONAL FACTOR IN BREAST CANCER.
Hilf R, Siiteri PK, Shellabarger, Korenman SG, Rosen, Nandi
Dept. Biochemistry and Univ. Rochester Cancer Center, Univ. Rochester, Rochester, NY, 14642
Banbury Rep; 8:317-337 1981

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

31. ONCOGENES.
Bishop JM
Univ. California, San Francisco, CA
Sci Am; 246(3):80-84,87-92,154 1982

32. HUMAN CANCERS AND HUMAN VIRUSES.
Waterson AP
Royal Postgraduate Medical Sch., London W12 0HS, England
Br Med J; 284(6314):446-448 1982

33. THE IMMUNOLOGY OF CANCER OF THE HEAD AND NECK WITH PARTICULAR REFERENCE TO ORAL CANCER.
Scully C
Univ. Dept. Oral Medicine and Pathology, Immunology Unit, Glasgow Dental Hosp., 378 Sauchiehall St., Glasgow G2 3JZ, Scotland
Oral Surg; 53(2):157-169 1982

METHODOLOGY IN CARCINOGENESIS RESEARCH

34. THE USE OF MAMMALIAN CELL MUTANTS TO STUDY THE MECHANISMS OF CARCINOGENESIS.
Trosko JE, Chang CC, Warren ST, Liu PK, Wade MH, Tsushimoto G
Dept. Pediatrics and Human Development, Michigan State Univ., Coll. Human Medicine, East Lansing, MI, 48824
Gann Monogr Cancer Res; 27:159-179 1982

35. BIOLOGICAL MONITORING IN THE IDENTIFICATION OF THE CANCER RISK OF INDIVIDUALS EXPOSED TO CHEMICAL CARCINOGENS.
Vainio H, Sorsa M, Rantanen J, Hemminki K, Aitio A
Inst. Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland
Scand J Work Environ Health; 7(4):241-251 1981

36. A CRITICAL REVIEW OF BIOLOGICAL METHODS FOR THE DETECTION OF FUNGAL TOXINS IN FOODS AND FOODSTUFFS.
Watson DH, Lindsay DG
Food Science Div., Ministry of Agriculture, Fisheries, and Food, Great Westminster House, Horseferry Road, London SW1P 2AE, England
J Sci Food Agric; 33(1):59-67 1982

37. PITUITARY AND STEROID HORMONES IN RADIATION-INDUCED MAMMARY TUMORS.
Shellabarger CJ
Medical Dept., Brookhaven National Lab., Upton, NY, 11973
Banbury Rep; 8:339-351 1981

38. ASPERGILLUS NIDULANS: SYSTEMS AND RESULTS OF TESTS FOR CHEMICAL INDUCTION OF MITOTIC SEGREGATION AND MUTATION. I. DIPLOID AND DUPLICATION ASSAY SYSTEMS. A REPORT OF THE U. S. EPA GENE-TOX PROGRAM.
Kafer E, Scott BR, Dorn GL, Stafford R
Dept. Biology, McGill Univ., 1205 Ave. Docteur Penfield, Montreal H3A 1B1, Canada
Mutat Res; 98(1):1-48 1982

OTHER REVIEWS IN CARCINOGENESIS

39. MATHEMATICAL MODELS OF THE CELL CYCLE WITH A VIEW TO TUMOR STUDIES.
Bertuzzi A, Gandolfi A, Giovenco MA
Centro di Studio dei Sistemi di Controllo e Calcolo Automatici del C.N.R., Via Eudossiana 18, 00184 Rome, Italy
Math Biosci; 53(3/4):159-188 1981

40. NEOPLASMS IN DOMESTIC ANIMALS: A REVIEW OF EXPERIMENTAL AND SPONTANEOUS CARCINOGENESIS.

Madewell BR

Section Clinical Oncology, Dept. Veterinary Surgery, Univ. California, Davis, CA, 95616

Yale J Biol Med; 54(2):111-125 1981

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SERIES CK03 No. 82/08 AUGUST, 1982

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In this issue:

- STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET..... 1-6
- EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET..... 7-8
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- GENERAL REVIEWS AND OTHER STUDIES..... 20-25

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

1. AN INDEPENDENT ANALYSIS OF THE NATIONAL CANCER INSTITUTE STUDY ON NON-NUTRITIVE SWEETENERS AND BLADDER CANCER.

Walker AM, Dreyer NA, Friedlander E, Loughlin J, Rothman KJ, Kohn HI
(c/o Dreyer), Epidemiology Resources, Inc., P. O. Box 57, Chestnut Hill, MA, 02167

Am J Public Health; 72(4):376-381 1982

We evaluated the possible relation between use of non-nutritive sweeteners and bladder cancer using data obtained from the National Cancer Institute Bladder Cancer Study under the Freedom of Information Act. In the general study group, there was no evidence for an association between non-nutritive

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sweeteners and bladder cancer. Control for a variety of factors through multivariate techniques diminished the plausibility of earlier interpretations of these data, which had raised the possibility that certain subgroups of users of non-nutritive sweeteners might be at an increased risk for bladder cancer. We found that the putative effects of non-nutritive sweeteners were not consistent among subgroups with similar baseline risk, did not display consistent dose-response trends, and were subject to considerable sampling error. We concluded that the data provided little evidence that non-nutritive sweeteners increase risk for bladder cancer among subgroups of users, and that definitive evidence on this question is beyond the reach of conventional research. (Author abstract) (2 Refs)

2. NON-NUTRITIVE SWEETENERS AND BLADDER CANCER.

Hoover R, Hartge P

Environmental Epidemiology Branch, NCI, NIH, Landow Building 3C07, Bethesda. MD, 20205

Am J Public Health; 72(4):382-383 1982

A recent report by Walker et al re-evaluating an NCI study revealed no association between bladder cancer risk and past consumption of artificial sweeteners in the total study population. Certain procedures of this study were criticized, including omission of design variables (sex and age) from generation of risk scores and statements concerning the role of epidemiology in assessing low-level risks. (5 Refs)

3. CALCULATION OF POTENTIAL BIAS IN THE ODDS RATIO ILLUSTRATED BY A STUDY OF SACCHARIN USE AND BLADDER CANCER.

Goldsmith DF

Dept. Applied Ecology, Res. Triangle Inst., P.O. Box 12194, Research Triangle Park, NC, 27709

Environ Res; 27(2):298-306 1982

Criticisms of questionnaire studies often emphasize methodological weaknesses which, if unexamined, may lead to biased risk estimates. Observational studies of the relationship between saccharin use and the risk of bladder cancer illustrate this point. This paper describes the potential biases due to misclassification and to the use of prevalent rather than incident cases in a recent report by I.I. Kessler and J.P. Clark (1978, J Amer Med Assoc 240, 349-355). Assuming the true risk ratio is greater than unity, nondifferential misclassification of exposure leads to an observed odds ratio or relative risk closer to the null value. Differential misclassification of exposure results in either an overestimate or an underestimate of the true risk. Varying the sensitivity and specificity of the exposure among cases and controls alters the risks ratios dramatically. The use of prevalent rather than incident cases in a hospital-based-control study does not affect the risk measure unless it can be demonstrated that exposure differentially influences the force of morbidity or mortality. The exclusion of a large number of deceased cases in Kessler and Clark's report may underestimate the risk ratio by 18-28%. Unless other factors are discovered, the identified biases will not meaningfully alter the conclusion that there is no association between saccharin use and bladder cancer in Kessler and Clark's study. (Author abstract) (15 Refs)

4. STUDIES ON THE ENVIRONMENTALLY INDUCED CONTAMINATION OF FOODS WITH CANCERIGENIC HYDROCARBONS IN GDR AND IN HUNGARIAN PEOPLE'S REPUBLIC.

Fritz W, Soos K

Zentralinstitut fur Ernahrung, Arthur Scheunert-Allee 114-116, DDR-1505 Bergholz-Rehbrucke, E. Germany

Nahrung; 25(10):905-913 1981

Environmentally induced contamination of foods with cancerigenic hydrocarbons was measured by determination of benzo[a]pyrene (BaP) in food grown close and far away from industrial plants (IP) in German Democratic Republic (GDR) and in Hungarian People's Republic (HPR). BaP content of vegetables grown far away from IP in GDR was 0.01-2.3 ug/kg of fresh wt and in HPR was 0.05-3.85 ug/kg with av values of 0.43 ug/kg and 0.6 ug/kg, respectively. The BaP content of vegetables grown close to IP in GDR was 0.15-12.4 ug/kg, and in HPR was 0.05-19.6 ug/kg. In HPR values obtained near a new petrochemical plant were 3.5 times higher than values obtained far away from IP; but values obtained near an old petochemical plant were 8 times higher. Degree of contamination depends very much on the type of industrial plant. Values obtained in GDR close to coking, tar extraction, or carbon black plants were 10-100 times higher. (30 Refs) (In GER)

5. COMPARATIVE STUDY OF CONTAMINATION OF PLANT FOOD PRODUCTS BY CARCINOGENIC POLYCYCLIC AROMATIC HYDROCARBONS (PAH) IN THE USSR AND GDR.

Shabad LM, Khesina AIa, Solenova LG, Dikun PP, Kalinina IA, Engst R, Fritz V

All-Union Cancer Res. Inst., Moscow, USSR

Vopr Pitan; (1):56-59 1982

The results of comparative evaluation of the concentrations of benzo(a)pyrene (BP) in the soil and in various agricultural plants in the Soviet Union and in the East Germany are reported. The increase of BP level in the soil from 0.5-1,000 ug/kg to greater than 15,000 ug/kg resulted in the increase of BP concentration from 0.4 ug/kg to 17.0 ug/kg in the wheat grain and from 2.0 ug/kg to 10.0 ug/kg in the sunflower seed. (11 Refs) (In RUS)

6. NATURAL OCCURRENCE OF MONILIFORMIN TOGETHER WITH DEOXYNIVALENOL AND ZEARALENONE IN TRANSKEIAN CORN.

Thiel PG, Meyer CJ, Marasas WF

Natl. Res. Inst. Nutritional Diseases, South African Medical Res. Council, Tygerberg 7505, Republic of South Africa

J Agric Food Chem; 30(2):308-312 1982

Fusarium infected ears of corn grown in a high human esophageal cancer incidence region were assayed for deoxynivalenol (DON), moniliformin (MF), zearalenone (ZL), T-2 toxin, and diacetoxyscirpenol. Cultures of F graminearum, F verticillioides, and F sacchari var subglutinans from the corn were raised in vitro and assayed for the chemicals. Only DON, MF, and ZL were found in the corn. F graminearum produced DON and ZL, F sacchari produced MF, and F verticillioides produced none. (24 Refs)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

7. IS COFFEE DRINKING A HEALTH HAZARD?

Chaudhuri MM, Ghosh S
Botany Dept., Bose Inst., Calcutta 700009, India
Sci Culture; 47(5):157-160 1981

Whether caffeine or its derivatives is a health hazard is discussed with respect to chemistry, metabolism, and mutagenicity testing. Evidence suggests that caffeine at low doses may act as a stimulator of the nervous system. High consumption, particularly in pathological conditions, may be hazardous. The dose of caffeine required for embryotoxicity is 100 mg/kg (equivalent to 40 cups of coffee). Mutagenicity testing has led to various conflicting results: caffeine has been found to be nonmutagenic, comutagenic, or a potent mutagen, particularly at high doses. (44 Refs)

8. CARCINOGENICITY EXAMINATION OF INFLORESCENCE OF ZINGIBER MIOGA ROSCOE.

Hirono I, Mori H, Kato K, Hosaka S, Aiso S
Dept. Carcinogenesis and Cancer Susceptibility, Inst. Medical Sciences, Univ. Tokyo, Minato-ku, Tokyo 108, Japan
Cancer Lett; 15(3):203-208 1982

Inflorescence of Zingiber mioga, a vegetable commonly used in Japan, was tested for carcinogenicity on oral administration in ACI rats (10 males, 10 females, 1-1.5 mo old) fed a mioga diet for 365 days and Fischer 344 rats (15 males, 15 females, 1.5 mo old) fed a mioga diet for 639 days. Eighteen of 20 ACI rats survived more than 336 days and 14 survived until the end of the experiment on day 480. The tumors found in rats are tabulated. One male and one female rat had urinary papilloma, and females had transitional cell carcinoma (TCC) of the urinary bladder (UB). In the control group one male developed interstitial cell tumor of the testis (ICTT) and one female TCC of the UB. A significant difference was observed between the incidence of UB tumors in female rats in the experimental and control groups. All Fischer 344 rats on the mioga diet survived for more than 391 days and 22/30 rats survived until the end of the experiment. No UB tumors were observed in the experimental or control group. ICTT and pituitary and lung adenomas were found in both the experimental and control group. No significant difference was observed in the incidence of these tumors. Three sc fibromas, one squamous cell carcinoma of the salivary glands, and three adrenal cortical adenomas were found in the control group. The incidences of these tumors were also not significantly different in the experimental and control groups. (9 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

9. RELATIONSHIP BETWEEN VITAMIN E AND CANCER - FACTS, NOT FANCY.

Ellison NM
Dept. Medical Oncology, Geisinger Medical Center, Danville, PA
Cancer Bull; 34(1):43-44 1982

The roles of vitamin E in inhibition of carcinogenesis and amelioration of conventional cancer treatment toxicity are discussed. Studies with mammalian models suggest that vitamin E may act as a protector against radiation-induced cellular damage and as a radiosensitizer in cancer treatment. The conflict between these antagonistic functions must be resolved. Vitamin E has been shown to ameliorate the toxic effects of doxorubicin in animal models without adversely affecting antineoplastic activity. Vitamin E may also act as an inhibitor of stomach cancer by inhibiting the reactions converting nitrites and nitrates to nitrosourea; possible prevention of gastric cancer by steady consumption of vitamin E and C has been suggested. Vitamin E, an antioxidant with the ability to inhibit free-radical-induced lipid peroxidation, may have some role in human carcinogenesis and cancer treatment, but evidence to support such a role is insufficient. (16 Refs)

10. PROGRESSION OF CARCINOGEN-INDUCED FOCI OF GAMMA-GLUTAMYLTRANSPEPTIDASE-POSITIVE HEPATOCYTES TO HEPATOMAS IN RATS FED A CHOLINE-DEFICIENT DIET.

Takahashi S, Lombardi B, Shinozuka H
(c/o Shinozuka) Dept. Pathology, Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA, 15261
Int J Cancer; 29(4):445-450 1982

Following previous findings that feeding a choline-deficient (CD) diet to rats strongly promotes the evolution of liver cells, initiated by a chemical carcinogen, to foci of gamma-glutamyltranspeptidase (gammaGT)-positive hepatocytes, we investigated whether a CD diet could also promote the evolution of gammaGT-positive foci to hepatomas. GammaGT-positive foci were induced in male Sprague-Dawley rats by administration of a single dose of diethylnitrosamine followed by 2 wk feeding on a CD diet containing 0.02% acetylaminofluorene. Immediately thereafter, one group of rats was fed a plain CD diet and the other a choline-supplemented (CS) diet, and subgroups of animals were killed periodically for analysis of the number and size of gammaGT-positive foci and development of hepatomas. During the first 7 wk after switching the diets, the number and size of the foci increased in both groups. At 12 and 16 wk, the number and size of foci began to decline in rats fed the CS diet. However, in the CD group, there was a progressive increase in the size with coalescence of the foci, as well as development of neoplastic nodules. These lesions were followed at 20 and 28 wk by development of hepatomas at a high incidence rate. In rats fed the CS diet, a few scattered gammaGT-positive foci, and small neoplastic nodules remained in the liver at 20 and 28 wk, but no hepatomas developed. These results show that the CD diet is a potent promoter of the evolution of foci of altered hepatocytes to hepatomas, and that most of the gammaGT-positive foci are reversible lesions. (Author abstract) (37 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

11. PRECANCEROUS CONDITIONS AND CANCER OF THE STOMACH.

Duris I, Koska M, Zavacic M, Mikulecky M, Holly D
Katedra internistickych disciplin, I Lekarska fakulta Univerzity Komenskeho, Mickiewiczoya 13, 88421 Bratislava, Czechoslovakia
Bratisl Lek Listy; 76(5):540-551 1981

Precancerous conditions and cancer of the stomach were studied in 4,159 patients subjected to gastroscopy. The histo-

logical diagnosis was that of gastric polyps in 112 patients, atrophic gastritis with intestinal metaplasia (AGIM) in 190, and gastric cancer in 103, including intramucosal carcinoma in 9. All patients with gastric carcinomas were in the age bracket 20-90 yr, and the frequency was higher among men than among women in all age groups. The incidence of AGIM was higher among women than among men in the age group 40-70 yr, especially in the age group 50-60 yr. Gastric cancer was found to occur at a significantly higher age among agricultural workers than in the other occupational groups. Concurrent disease as risk factor was found in 47% of 42 patients with gastric xanthomas. Gastric stump carcinoma was found in 23% of the patients subjected to gastric resection for gastric or duodenal ulcer. Forty-two parameters were studied in the gastric cancer patients and in patients without this disease by the pattern recognition method. Consumption of garlic, onions, home made smoked meat, as well as persistent emotion of fear were found to be linked most closely with gastric cancer. Restriction of burnt food and consumption of generous amounts of fruits and vegetables appear to be beneficial against gastric cancer. (41 Refs) (In SLO)

12. STUDIES ON POTENTIAL CARCINOGENS IN THE DIET OF INDIVIDUALS AT HIGH RISK FOR ESOPHAGEAL CANCER.

Li MH
Inst. Cancer Res., Chinese Acad. Medical Sciences, Beijing, People's Republic of China
Cancer Research in the People's Republic of China and the United States of America: Epidemiology, Causation and New Approaches to Therapy, Proceedings of a Conference held in New York, 28-29, March, 1980. Marks PA, ed. New York, Grune and Stratton, 273 pp., 1981.

Studies on etiological agents of esophageal cancer in the high-risk areas are discussed. Environmental factors apparently play an important role in the development of esophageal cancer. Some foodstuffs in Linxian are frequently contaminated by Fusarium moniliforme and other fungi. Formation of dimethylnitrosamine, diethylnitrosamine, methylbenzylnitrosamine, and a new volatile N-nitroso compound, N-1-methylacetonyl-N-3-methylbutylnitrosamine has been demonstrated in moldy cornbread after an 8-day incubation, when a small amount of $NaNO_2$ was added initially. Other contributing factors in the environment are nutritional deficiencies in animal protein and fat, vitamins A, B2, and C, high levels of nitrates and nitrites in water and food, low molybdenum and zinc, imbalance of trace elements, and indoor air pollution from coal stoves during the winter. (10 Refs)

13. DIETARY WHEAT BRAN LOWERS COLONIC PH IN RATS.

Jacobs LR, Lupton JR
Dept. Internal Medicine, Sch. Medicine, Univ. California, Davis, CA, 95616
J Nutr; 112(3):592-594 1982

The effect of chronic dietary wheat bran supplementation on the pH of gastrointestinal contents was examined in 31 Sprague-Dawley rats. Ten control rats received a fiber-free diet while 21 rats were fed the same diet plus a 20% wheat bran supplement. The fiber-fed rats developed a greater acidity of intestinal contents: cecal pH was 6.30 +-0.07 (SEM) as compared to 6.76 +-0.06 in the control group (p less than 0.001). The effect on pH of gastric contents was the opposite, 3.90 +-0.15 in the fiber-fed rats compared to 2.74 +-0.33 in the controls (p less than 0.01). These changes in gastrointestinal pH may be of relevance to the nutrient modulation of gastric and colonic tumor development. (Author abstract) (22 Refs)

14. DIET AND HORMONE LEVELS IN SEVENTH-DAY ADVENTIST TEENAGE GIRLS.

Gray GE, Williams P, Gerkins V, Brown JB, Armstrong B, Phillips R, Casagrande JT, Pike MC, Henderson BE
(c/o Henderson) Dept. Family and Preventive Medicine, Univ. Southern California, 2025 Zonal Ave., Los Angeles, CA, 90033
Prev Med; 11(1):103-107 1982

Differences in age at menarche and in plasma and urine hormone levels between meat eating and non-meat-eating Adventists were investigated. White girls (14-17 yr old) who had menstrual cycles 27-32 days in length, had never used oral contraceptives, had never been pregnant, did not take medication regularly, had no prior history of gynecological, endocrine, or neoplastic diseases, and were willing to have blood and urine specimens taken for hormone analysis were included in the study. Twenty-three girls who had never eaten meat on a regular basis (VE) and 26 who were eating meat, fish, or poultry at least weekly (NVE) completed a detailed questionnaire on dietary habits. Blood samples were collected between 30 min and 2 hr after arising on days 11 and 22 of the same menstrual cycle. VE and NVE girls did not differ in height, wt, or age at menarche. The relative amounts of protein, fat, and carbohydrates expressed as percentages of calories consumed by both groups were similar. The hormone levels were similar and no statistically significant differences were found. Risk from dying of breast cancer for California Seventh-Day Adventist women is approx 30% lower than that of the general population; the difference has been attributed to dietary habits. The hypothesis that meat consumption is a risk factor for breast cancer is questioned. (19 Refs)

15. DIET AND HORMONE PROFILES IN TEENAGE GIRLS IN FOUR COUNTRIES AT DIFFERENT RISK FOR BREAST CANCER.

Gray GE, Pike MC, Hirayama T, Tellez J, Gerkins V, Brown JB, Casagrande JT, Henderson BE
(c/o Henderson) Dept. Family and Preventive Medicine, Univ. Southern California Sch. Medicine, 2025 Zonal Ave., Los Angeles, CA, 90033
Prev Med; 11(1):108-113 1982

Findings on diet and possible breast cancer risk factors in adolescent girls in Chile (CH), Japan (JA), Papua New Guinea (PNG), and the United States (US) are presented. The 51 CH girls (mean age 17.0 yr), 50 JA girls (mean age 16.9 yr), and 68 PNG girls (mean age 17.7 yr) were from rural areas, and the 50 US girls (mean age 16.5) were all members of the same church. The subjects were postmenarchial with an av cycle of 26-32 days and had never been pregnant or used oral contraceptives or other medication known to influence sex hormone levels (SHL). Blood samples were to be collected in the morning not less than 30 min after the subject awakened on day 11 of the menstrual cycle; the condition was achieved for all girls except for 32% of CH girls. Obvious differences in body build among the subjects from the countries were noted. Dietary data and mean SHL are presented. The only significant differences were in urinary estriol and the urinary estriol ratio. The levels were considerably higher in PNG girls than in those from girls from the other three countries. Correlation coefficients were calculated to assess the relationship between anthropometric measurements, age, age at menarche, percentage of calories from fat, carbohydrate, and protein, and SHL within each country. No consistent relationship between SHL and any of the other factors were found. The anthropometric measurements and age at menarche were not related to the dietary variables within each country. According to the results, at least with diets leading to the same age at menarche, an in-

creased percentage of calories from fat does not increase prolactin levels. (24 Refs)

16. NUTRITION AND CANCER: MECHANISMS OF GENOTOXIC AND EPIGENETIC CARCINOGENS IN NUTRITIONAL CARCINOGENESIS.

Weisburger JH, Horn C
American Health Foundation, Valhalla, NY, 10595
Bull NY Acad Med; 58(3):296-312 1982

Mechanisms of carcinogenesis are reviewed with regard to type of chemical carcinogen - genotoxic, cocarcinogen, or promoter. Examples and mode of action are given for each. The role of epigenetic agents in nutritionally linked cancers, particularly large bowel or colon cancer, is discussed; specific dietary elements considered relevant through studies in human and animal models are the amount of dietary fat and fiber and mode of cooking. The operation of these elements is through epigenetic mechanisms, which are dose and time dependent. A reduction in dose should lead to lowering of risk, which may eventually be applied to humans through dietary adjuvant therapy. (71 Refs)

17. EARLY CHANGES IN MITOCHONDRIAL MONOAMINE OXIDASE ACTIVITY OF RAT LIVER DURING HEPATOCARCINOGENS FEEDING.

Kuroiwa Y, Yoshida T, Yamamoto T
Dept. Biochemical Toxicology, Sch. Pharmaceutical Sciences, Showa Univ., 1-5-8 Hatanodai Shinagawa-ku, Tokyo 142, Japan
Excerpta Med Int Congr Ser; 564():241-250 1982

The effects of 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB), 2-acetylamino-fluorene (2-AAF), 2-aminofluorene (2-AF), 2-nitrofluorene (2-NF), and N-hydroxy-2-AAF on mitochondrial monoamine oxidase (MAO) activity of rat liver are reported. Male and female Donry rats were used. Significant decrease (70%) in MAO-B activity was observed during 3'-Me-DAB feeding. The changed patterns of MAO-B activity are similar to those of rat hepatoma. When rats were fed a diet containing 0.05% 2-AAF, a remarkable decrease of MAO-B activity was observed, while MAO-A activity was not affected. In female rats fed 0.05% 2-AAF diet, MAO-B activity was not changed after 1 wk, but decreased to some extent at 3 and 12 wk after the exposure to the carcinogen. The intensity of the MAO-B activity decrease in female rats was to a lesser extent as compared to that of male rats at each time period. 2-AAF higher concentrations inhibited MAO-B activity when added in vitro, but MAO-A activity was strongly inhibited by addition of 10(-7) M 2-AAF. Each compound of fluorene derivatives tested was administered po at a dose of 200 micro moles/kg daily for 7 days. N-hydroxy-2-AAF and 2-AF decreased MAO-B activity. 2-NF did not show any effect under experimental conditions. Effect of 2-AAF on MAO-B activity was examined in thyroxine-treated and riboflavin-deficient rats. An additive decrease of MAO-B activity was observed in 2-AAF and thyroxine-treated animals as compared to the decrease produced by the separate administration. When rats were fed riboflavin-deficient diet for 2 wk, MAO-B activity was decreased to 60% of the control levels. The decreased activity tended to return to control levels if rats were fed the basal diet again. (27 Refs)

18. IN VIVO STABILITY OF NITRITE AND NITROSAMINE FORMATION IN THE DOG STOMACH: EFFECT OF NITRITE AND AMINE CONCENTRATION AND OF ASCORBIC ACID.

Lintas C, Clark A, Fox J, Tannenbaum SR, Newberne PM
Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA, 02139
Carcinogenesis; 3(2):161-165 1982

Formation of N-nitrosodimethylamine (NDMA) from the precursors nitrite and dimethylamine has been studied in the chronically fistulated dog stomach. Measurements have also been made as a function of time for nitrite stability and gastric pH, and a nonabsorbable marker has been used to correct for gastric emptying and dilution. In the fasting animal NDMA forms within minutes after addition of the reactants if the pH is below 5. The presence of food in the stomach slows the reaction, and the additional presence of ascorbic acid greatly depresses the amount of NDMA formed. The concentration of nitrite in gastric juice declines rapidly after its introduction, but the concentration of NDMA declines even more rapidly than nitrite. This suggests that NDMA is probably rapidly absorbed directly from the dog stomach, in contrast to earlier results in rodent experiments. (Author abstract) (16 Refs)

19. INDUCTION OF ORNITHINE DECARBOXYLASE IN COLON AND LIVER BY STARVATION AND REFEEDING: A COMPARISON OF EFFECTS ON TOTAL AND HOLOENZYME.

Stanley BA, Kazarinoff M
Div. Nutritional Sciences and Section Biochemistry, Molecular and Cell Biology, Div. Biological Sciences Cornell Univ., Ithaca, NY, 14853
Biochem Biophys Res Commun; 105(2):773-777 1982

Short-term starvation and refeeding induced both the total and holo-ornithine decarboxylase (ODC) levels in the colon of rats. After brief starvation alone, a significant decrease in total ODC was observed in the colon; there was no effect on colon holo-ODC. Similar effects on total ODC level were seen in the liver; neither starvation-refeeding nor starvation alone had any effect on liver holo-ODC levels. This is the first demonstration of the dietary induction of ODC in the colon. It is of interest to investigate whether this induction simply reflects a compensatory increase in general protein synthesis following starvation, or whether it represents a specific induction such as that observed during tumor promotion by chemical carcinogens. (18 Refs)

GENERAL REVIEWS AND OTHER STUDIES

20. ASSESSING THE VALUE OF MEGANUTRIENTS IN DISEASE.

DiPalma JR, McMichael R
Dept. Pharmacology, Hahnemann Medical Coll. and Hosp., Philadelphia, PA
Bull NY Acad Med; 58(3):254-262 1982

The so called mega doses of vitamins are in fact only large doses and probably constitute a range which may be termed a pharmacological dose. Some vitamins do have an accurate pharmacological dose for a specific indication. A good example is thiamine, which, in a dose about 20x the human parenteral dose of 100 mg, will, in cats, cause neuromuscular and gang-

lionic block. This is because the chemistry of thiamine is such that it is capable of attachment to cholinergic receptors. The vitamin function which pertains to cofactor function to a particular enzyme is distinct and can be separated from the pharmacological dose which involves a different receptor. Various therapies have been advocated based on pharmacological doses of vitamins such as the nicotinic acid therapy of hyperlipemia, niacin therapy for mental disease, and vitamin C treatment for the common cold and cancer. None have been entirely successful and the benefit to be gained is for the most part overshadowed by the risk and discomfort of therapy. This is especially true of the niacin therapy of mental illness with its serious risk of liver toxicity. Vitamin C therapy of the common cold has been thoroughly investigated by large scale tests and is discredited. Vitamin C therapy of cancer is also questionable but a good amount of experimental data relates Vitamin C to cancer and suggests further investigation. None of the mega therapies have substantive experimental data to substantiate their claims. Most data advanced are clinical testimonials unacceptable in an age where precision can be obtained in dose-response situations even in the clinic. (Author abstract) (26 Refs)

21. ASSESSING HUMAN EPIDEMIOLOGIC DATA ON DIET AS AN ETIOLOGIC FACTOR IN CANCER DEVELOPMENT.

Enstrom JE
Sch. Public Health, Univ. California, Los Angeles, CA
Bull NY Acad Med; 58(3):313-322 1982

Assessment of the relationship between diet and cancer is reviewed. Epidemiology of cancer among human populations consists of randomized, controlled trials based on interpretation of naturally occurring phenomena. The strongest epidemiologic data relating diet to cancer are geographic correlations between such factors as per capita fat consumption and age adjusted colon and breast cancer rates. Weaknesses exist in such correlation: large variation in colon cancer rates occurs within the United States (US), even when fat intake remains constant. Studies of Japanese who migrate from Japan to the US provide another form of epidemiologic evidence for a correlation between diet and cancer. Japanese emigrants in the US have increased rates for colon and breast cancer as compared to native Japanese; such selective use of epidemiologic data is partially contradicted by other data which indicate that the overall total cancer rate among Japanese Americans remains unchanged, with a total death rate lower than that of native Japanese. Specific populations which maintain diets different from the average American diet are discussed, using as examples Mormons and Mexican Americans. No data has demonstrated any specific relationship between diet and cancer rate. (26 Refs)

22. DIETARY FIBER.

Kay RM
Dept. Surgery, Toronto Western Hosp., Univ. Toronto, Toronto M5T 2S8, Canada
J Lipid Res; 23(2):221-242 1982

Dietary fiber is plant-derived material that is resistant to digestion by human alimentary enzymes. Fiber may be divided into two broad chemical classes: (1) non-alpha-glucan polysaccharides (cellulose, hemicelluloses, and pectins) and (2) lignins. Dietary fiber behaves within the gastrointestinal tract as a polymer matrix with variable physicochemical properties including susceptibility to bacterial fermentation, water-holding capacity, cation-exchange, and adsorptive functions. These properties determine physiological actions of fiber and are dependent on the physical and chemical composition of the fiber. Fiber undergoes compositional changes as a consequence of bacterial enzymatic action in the colon. Dietary fiber is of clinical significance in certain disorders of colonic function and in glucose and lipid metabolism. Dietary fiber increases stool bulk by acting as a vehicle for fecal water and by increasing fecal bacterial volume. Use of fiber in the treatment of constipation and uncomplicated diverticular disease is well established. By increasing stool bulk, fiber also reduces the fecal concentration of bile acids and other substances. Certain types of fiber decrease the rate of glucose absorption and attenuate postprandial rises in blood glucose and insulin. Plasma cholesterol levels are reduced by mucilaginous forms of fiber. This effect appears to be mediated in part by an increase in fecal acidic sterol excretion. (Author abstract) (266 Refs)

23. DIETARY FIBER AND DISEASE.

Kritchevsky D
Wistar Inst. Anatomy and Biology, Philadelphia, PA
Bull NY Acad Med; 58(3):230-241 1982

The association between dietary fiber and disease is reviewed. Classification of fiber is based on chemical components of the main chain; various fiber classes (pectins, hemicellulose, cellulose, lignin) have different physiological actions in humans. The possible association between levels of dietary fiber and colon cancer and atherosclerosis is discussed. A diet high in complex carbohydrates (high fiber diet) has resulted in reduced serum cholesterol and glucose and also reduced insulin requirements. No directly positive influence of dietary fiber on lipid metabolism and colon cancer has consistently been found. (77 Refs)

24. FIBRES, NUTRITION AND THE GUT.

Schrijver M, Tytgat GN
Dept. Internal Medicine, Div. Gastroenterology, Univ. Amsterdam, 1 e Helmerstraat 104, 1054 EG Amsterdam, The Netherlands
Neth J Med; 25(2):49-55 1982

The constitution of dietary fiber and its normal function and fate in the gastrointestinal tract are reviewed. Potential for carcinoma of the colon and rectum and other diseases as a result of fiber deficiency is discussed. Conducting and/or interpretting most studies is complicated by both the nonstandard nature of 'fiber' and its interactions with intestinal microflora and bile. (70 Refs)

25. IS AFLATOXIN B1 A HEPATIC CARCINOGEN IN MAN?.

Franco D, Castaing D, Brechnot C, Morin J
Groupe de Recherche de Chirurgie Hepatique (INSERM U-17), Hopital Paul-Brousse, 14, avenue Paul-Vaillant-Couturier, F-94800 Villejuif, France
Gastroenterol Clin Biol; 6(2):125-128 1982

Arguments for the hepatic carcinogenicity of aflatoxin B1 (AFB) in man are discussed: AFB is a carcinogen in nonhuman primates; in vitro studies show that the metabolism of AFB by human liver microsomes is identical to that observed in rat microsomes; AFB liver toxicity in man has been well-demonstrated; epidemiologic studies have shown that hepatocellular carcinoma is particularly frequent in countries with high levels of AFB contamination of food. (32 Refs) (In FRE)

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CHEMICAL CARCINOGENESIS

1. TOXICITY AND METABOLISM OF ACRYLONITRILE.
Sapota A, Chmielnicka J
Zaklad Metabolizmu Substancji Toksycznych, Instytut Medycyny Pracy, ul. Teresy 8, 90-950 Lodz, Poland
Med Pr; 32(1):25-33 1981
In POL

2. A REVIEW OF THE GENOTOXICITY OF FOOD, DRUG AND COSMETIC COLOURS AND OTHER AZO, TRIPHENYLMETHANE AND XANTHENE DYES.
Combes RD, Haveland-Smith RB
Dept. Biological Sciences, Portsmouth Polytechnic, King Henry I St., Portsmouth, PO1 2DY, Hants, England
Mutat Res; 98(2):101-248 1982

3. CARCINOGENICITY OF AIRBORNE FINE PARTICULATE BENZO(A)PYRENE: AN APPRAISAL OF THE EVIDENCE AND THE NEED FOR CONTROL.
Perera F
Natl. Resources Defense Council, Inc., 122 East 42nd St., New York, NY, 10168
Environ Health Perspect; 42:163-185 1981

4. TOXICOLOGY OF N-NITROSO COMPOUNDS.
Coulston F, Olajos EJ
Albany Medical Coll., Albany, NY, 12208
Ecotoxicol Environ Saf; 6(1):89-96 1982

5. CRITICAL REVIEW OF THE MUTAGENIC AND OTHER GENOTOXIC EFFECTS OF DIRECT COAL LIQUEFACTION.
Kimball RF, Munro NB
Oak Ridge Natl. Lab., Oak Ridge, TN
Critical Review of the Mutagenic and Other Genotoxic Effects of Direct Coal Liquefaction. Available from National Technical Information Service, Springfield, VA, as DE81027859:, 216 pp., 1981.

6. CARCINOGENICITY AND ITS EFFECT ON MEDICINAL RESEARCH PROGRAMS IN THE USA.
Van Ryzin RJ
Pharmaceutical Res. and Development, Sandoz, Inc., East Hanover, NJ, 07936
Med Res Rev; 2(2):153-166 1982

7. ANTIPROTOZOAL DRUGS (SIDE EFFECTS).
Nir I
Dept. Pharmacology and Experimental Therapeutics The Hebrew Univ., Hadassah Medical Sch., Jerusalem, Israel
Side Eff Drugs Annu; (5):278-284 1981

8. DRUGS USED IN TUBERCULOSIS AND LEPROSY (SIDE EFFECTS).
Elis J
Inst. Pharmacology, Czechoslovak Acad. Sciences, Albertov 4, Prague 2, Czechoslovakia
Side Eff Drugs Annu; (5):290-297 1981

9. SEX HORMONES AND RELATED COMPOUNDS, INCLUDING ORAL CONTRACEPTIVES.
Plotz EJ, Nocke W, Schander K, Patt V, Kaulhausen H
Dept. Obstetrics and Gynecology, Univ. Bonn, 5300 Bonn-Venusberg, W. Germany
Side Eff Drug Annu; (5):358-380 1981

RADIATION CARCINOGENESIS

10. NEPTUNIUM - THE NEGLECTED ACTINIDE: A REVIEW OF THE BIOLOGICAL AND ENVIRONMENTAL LITERATURE.
Thompson RC
Biology Dept., Pacific Northwest Lab., Richland, WA, 99352
Radiat Res; 90(1):1-32 1982

11. CARCINOGENIC EFFECT OF RADIOACTIVE PRODUCTS.
Vasilenko Ila
No affiliation given
Vopr Onkol; 28(1):76-83 1982
In RUS

DIETARY FACTORS IN CARCINOGENESIS

12. FIBRES, NUTRITION AND THE GUT.
Schrijver M, Tytgat GN
Dept. Internal Medicine, Div. Gastroenterology, Univ. Amsterdam, 1 e Helmerstraat 104, 1054 EG Amsterdam, The Netherlands
Neth J Med; 25(2):49-55 1982

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EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

16. THE AH LOCUS, A MULTIGENE FAMILY NECESSARY FOR SURVIVAL IN A CHEMICALLY ADVERSE ENVIRONMENT: COMPARISON WITH THE IMMUNE SYSTEM.
Nebert DW
Developmental Pharmacology Branch, National Inst. Child Health, NIH, Bethesda, MD, 20205
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DIETARY ASPECTS OF CARCINOGENESIS

SERIES CK03 No. 82/09

SEPTEMBER, 1982

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STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. LIVER DNA BOUND IN VIVO WITH AFLATOXIN B1 AS A MEASURE OF HEPATOCARCINOMA INITIATION IN RAINBOW TROUT.

Whitham M, Nixon JE, Sinnhuber RO
(c/o Nixon) Dept. Food Science and Technology, Oregon State Univ., Corvallis, OR, 55427
JNCI; 68(4):623-628 1982

The in vivo binding of aflatoxin B1 (AFB1) to liver DNA was measured in rainbow trout subjected to various regimens and in coho salmon to determine if binding would correlate with tumor incidence and initiation of hepatocellular carcino-

HE 20.3173/2:

ma. Establishment of time-course binding parameters showed no significant difference between 4, 12, 24, or 48 hr. Doses of 5, 25, 100, and 300 ug AFB1/kg body wt produced an almost linear increase in binding with increasing dose. Compared to that of controls, AFB1-DNA binding was 2-fold lower in beta-naphthoflavone-treated trout and 20-fold lower in coho salmon. These two comparisons reflected a decrease in the effective activation and binding of AFB1, correlated with tumor incidence, and are believed to correlate with a decrease in the initiation of hepatocellular carcinoma. AFB1 binding was not significantly altered by dietary protein or cyclopropenoid fatty acids, which are thought to increase hepatocellular carcinoma incidence through promotional effects. The data were consistent with the view that in vivo binding of AFB1 to liver DNA is a measure of cancer initiation by AFB1 and correlated with the incidence of hepatocellular carcinoma in salmonid fish. (Author abstract) (52 Refs)

2. THE BIOLOGICAL EFFECTS OF CARRAGEENAN AND OTHER SULFATED POLYSACCHARIDES WITH PARTICULAR ATTENTION TO IMMUNOLOGICAL AND CARCINOGENIC PROPERTIES.

Larsen B

Kraeftforskningsinstituttet, DK-8000 Arhus C, Denmark

Ugeskr Laeger; 144(12):860-863 1982

A review of studies on the biological effects of carrageenan shows that induction of colorectal cancer was observed in rats following long-term oral administration of degraded carrageenan. Native (high-molecular-weight) is not known to possess carcinogenic properties. Carrageenan stimulates tumor growth secondarily. The cocarcinogenic effect of carrageenan may be due to its immunosuppressive properties (toxic effect on macrophages and activation of macrophage suppressor cells). (29 Refs) (In DAN)

3. RELATIONSHIP AMONG STOMACH CANCER, NITRATE IN DRINKING WATER AND TRACE ELEMENTS (MEETING ABSTRACT)

Juhasz L, Hill ML, Nagy G, Vass A

Cancer Registry of County Szabolcs-Szatmar, Nyiregyhaza, Hungary

VI Meeting of the European Association for Cancer Research, October 12-15, 1981, Budapest, Hungary. The European Association for Cancer Research 275 pp., 1981.

According to the tabulation of Segi et al (1979) Hungary has one of the highest mortality rates from stomach cancer. There have been a number of studies concerning possible relationship between incidence of stomach cancer and high levels of nitrate in drinking water. Shallow-well waters in County Szabolcs-Szatmar, Hungary contained very often high nitrate levels. An epidemiological study was started in 1975 in the County Szabolcs-Szatmar to study these relationship. In more than 60% of localities was found high incidence of stomach cancer (over 20/100,000). In near to 90% of localities were found high nitrate in drinking water (over 100 mg/liter). High nitrate content in the walls was observed in 62% of the places with high stomach cancer incidence. Places located on cast and clay soil have significantly higher incidence of stomach cancer than the localities on sandy soil. According to nitrate level and stomach cancer incidence 420 urine samples were taken and analyzed in London. Heavy metals are industrial toxins and carcinogens. This year we began the analyses of some trace elements of the soil (Mg, Zn, Cu, Mn). (no Refs)

4. PASSAGE OF (14C)-AFLATOXIN B1 AND METABOLITES INTO RAT MILK.

Schroeder T, Carden BM, Luthy J, Schlatter C

Kant. Laboratorium, Kannenfeldstrasse 2, Postfach, CH-4012 Basel, Switzerland

Mitt Geb Lebensmitteluners Hyg; 73(1):57-70 1982

The passage of aflatoxin B1 (ATB1) from the digestive tract into milk was studied in two lactating rats. Rat 1 received 1.52 uCi (14C)-ATB1 po after giving birth and was milked 1, 4, 24, and 48 hr after administration of the radioisotope. Rat 2 received 1.94 uCi po after lactation wk 2 and was milked 2, 5, 24, and 48 hr afterwards. Levels of labeled ATB1 and the metabolites aflatoxin M1 (ATM1), aflatoxin B2a (ATB2a), and aflatoxicol (ATL) were measured in the milk of both rats and blood from the tail vein of rat 2. Specific activities in milk and blood decreased from 5,000 disintegrations/min/ml (dpm/ml) after 1 hr (rat 1) or 2 hr (rat2) to 2,000 dpm/ml at 4 hr (rat 1) or 5 hr (rat 2) and 1,000 dpm/ml at 24 hr (both rats). After fractionation of milk collected after i hr, the acetone-precipitated macromolecular phase contained 3.8% of the radioactivity, the aqueous phase 2.2%, the hexane phase 3.0%, and the dichloromethane (DCM) phase 86.9% after 1 hr. In milk collected after 4 hr the respective values for the different phases were 16.8%, 3.4%, 2.9%, and 58.6%. Analysis of the DCM extract by isotope dilution and thin-layer chromatography indicated that ATM1 accounted for 67.4% of the total radioactivity at 1 hr and 68.4 % at 4 hr. ATB1 accounted for 5.95% at 1 hr and 3.5% at 4 hr. ATB2a accounted for 0.8% of the total radioactivity. ATL accounted for 0%. Absence of ATL in rat milk may have been due to reoxidation of ATL by the liver. (49 Refs) (In GER)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

5. INTRA-UTERINE EXPOSURE TO SACCHARINE AND RISK OF BLADDER CANCER IN MAN.

Jensen OM, Kamby C

Danish Cancer Registry, Strandboulevarden 49, DK-2100 Copenhagen 0, Denmark

Int J Cancer; 29(5):507-509 1982

Animal experiments show that rats fed saccharine are more likely to develop bladder tumors if they have been exposed to saccharine in utero through their mothers' food. The risk of bladder tumors in humans following in utero exposure to saccharine has not been evaluated previously. In Denmark the use of saccharine increased sharply during the second World War as a result of scarcity of sugar, and import and export figures indicate that saccharine consumption was on av 4-5x higher during the war-time period than the pre-war decade. The risk of bladder tumors at ages 20-34 was 1.0 (95% confidence interval 0.7-1.6) among men born in 1941-1945 compared with men born 1931-1940. Among women the risk was 0.3 (0.1-1.0). This study provides no evidence of an increased risk of human bladder cancer during the first 30-35 yr of life associated with in utero saccharine exposure. (Author abstract) (20 Refs)

6. ARTIFICIAL SWEETENERS AND BLADDER CANCER IN MANCHESTER, U.K., AND NAGOYA, JAPAN.

Morrison AS, Verhoek WG, Leck I, Aoki K, Ohno Y, Obata K
Dept. Epidemiology, Harvard Sch. Public Health, Boston, MA
Br J Cancer; 45(3):332-336 1982

We have evaluated the relation between cancer of the lower urinary tract ('bladder cancer') and the use of artificial sweetners, by means of case-control studies in Manchester, UK, and Nagoya, Japan, areas where extensive use occurred 30-40 yr ago. In each area, a broadly based series of cases (555 in Manchester, 293 in Nagoya) was interviewed and a series of controls (735 in Manchester, 589 in Nagoya) chosen from the general population. A history of use of sugar substitutes primarily saccharin, was not associated with an elevated risk of bladder cancer in either study area. Risk of bladder cancer did not increase regularly with frequency or duration of use of sugar substitutes. Data on dietetic beverages were not obtained in Nagoya. This exposure was not associated with a greater risk of bladder cancer in Manchester. The results of this study suggest that use of artificial sweetners confers little or no risk of bladder cancer. (Author abstract) (10 Refs)

7. ORGAN-SPECIFIC PROMOTING EFFECTS OF PHENOBARBITAL SODIUM AND SODIUM SACCHARIN IN THE INDUCTION OF LIVER AND URINARY BLADDER TUMORS IN MALE F344 RATS.

Nakanishi K, Fukushima S, Hagiwara A, Tamano S, Ito N
First Dept. Pathology, Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizucho-cho, Mizuho-ku, Nagoya 467, Japan
JNCI; 68(3):497-500 1982

Studies were conducted to determine whether initial treatment with N-2-fluorenylacetamide (2-FAA) or N-butyl-N-(4-hydroxybutyl)nitrosamine (BBN) followed by treatment with either phenobarbital sodium (PB) or sodium saccharin (SS) induced or enhanced liver and/or bladder tumorigenesis. Male inbred F344 rats were given 0.05% PB and 5.0% SS in the diet for 32 wk after 4-wk administration of 0.02% 2-FAA or 0.01% BBN. PB significantly enhanced hepatocarcinogenesis after 2-FAA treatment and induced hyperplastic liver nodules after BBN treatment. The SS significantly enhanced induction of bladder hyperplasias, but not papilloma after BBN treatment, and induced bladder hyperplasias after 2-FAA treatment. However, PB had no effect on the bladder and SS had no effect on the liver after either 2-FAA or BBN treatment. The data indicate that although 2-FAA and BBN have tumor-initiating effects in the liver and urinary bladder, the tumor-promoting effects of PB and SS are organ-specific. The tumor-promoting effect of PB for the liver is relatively greater than that of SS for the urinary bladder. (Author abstract) (25 Refs)

8. CARCINOGENESIS BIOASSAY OF FD AND C YELLOW NO. 6 (CAS NO. 2783-94-0).

National Toxicology Program
P. O. Box 12233, Research Triangle Park, NC, 27709
Natl Toxicol Program Tech Rep Ser (208): 133 pp., 1981.

Carcinogenicity of FD and C No 6, (FDCY) a widely used food colorant, was investigated in 50 male and 50 female F344 rats and B6C3F1 mice fed diets containing 12,500 or 25,000 ppm FDCY for 103 wk. Groups of 90 male and 90 female rats and 50 male and 50 female mice were undosed controls. Mean body wt of high-dose male rats and high-dose male and female mice were slightly lower than those of controls. Hepatocellular carcinomas occurred in low-dose male mice at an incidence significantly higher than that in controls; however, since the incidence of these tumors in the high-dose group was not significantly higher than that in controls, and because of the variability of the occurrence of liver tumors in control mice, the increase was not clearly related to FDCY administration. Under the conditions used, no clear evidence of carcinogenicity of FDCY in F344 rats and B6C3F1 mice of either sex was found. (35 Refs)

9. DIFFERENTIAL EFFECTS OF DIETARY ANTIOXIDANTS OF CHEMICAL CARCINOGENESIS (MEETING ABSTRACT)

King MM, McCay PB
Oklahoma Medical Research Foundation, Oklahoma City, OK, 73104
Fed Proc; 41(3):358 1982

Female weanling Sprague-Dawley rats were fed a defined semipurified diet with antioxidants being supplemented throughout the experiments as indicated. In one set of experiments, the diets were supplemented with butylated hydroxytoluene (BHT). The rats were given a 10 mg oral dose of 7,12-dimethylbenz(a)anthracene at 50 days of age. The incidence of mammary adenocarcinomas was markedly lower in the BHT-supplemented rats compared to rats fed the same diet without BHT. When butylated hydroxyanisole (BHA) was supplemented in the diet instead of BHT, no inhibition of mammary tumor development was observed. In similar experiments, it was found that propyl gallate supplementation also offers protection against DMBA-induced tumorigenesis while alpha-tocopherol does not. These results raise the question: is the antioxidant property of the supplement actually related to inhibition of tumorigenesis or is it some other property of particular antioxidants (in this case, BHT and propyl gallate)? To determine if BHT may possibly be affecting some metabolic system associated with the activation of DMBA to the ultimate carcinogen, the effect of BHT supplementation on tumor induction was compared between a direct acting mammary carcinogen (nitrosomethylurea) and DMBA, which does require metabolic activation. BHT inhibited DMBA- but not NMU-induced mammary tumorigenesis, suggesting that the influence of BHT may involve inhibition of DMBA activation. (no Refs)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

10. CULTIVATED MUSHROOM MYCOTOXINS: CARCINOGENESIS AND CHEMISTRY (MEETING ABSTRACT)

Toth B, Ross A
University of Nebraska Medical Center, Omaha, NE, 68105
VI Meeting of the European Association for Cancer Research, October 12-15, 1981, Budapest, Hungary. The European Association for Cancer Research 275 pp., 1981.

Agaricus bisporus, the commonly eaten cultivated mushroom of commerce in the Western Hemisphere, contains beta-N-[gamma-L-(+)-glutamyl]-4 hydroxymethylphenylhydrazine (synonym: agaritine, A), its breakdown product 4-hydroxymethylphenylhydrazine (4-HMPH), and 4-(hydroxymethyl)benzenediazonium ion (4-HMBD), etc. It has also been shown in vitro that 4-HMPH yields to 4-methylphenylhydrazine (4-MPH). Carcinogenesis investigations have been performed with these four chemicals in Swiss mice in groups of 100 animals each. Two chemicals in this series, the

hydrochloride form of 4-MPH and the N'-acetyl derivative of 4-HMPH, induced tumors of lungs and blood vessels. In addition, the tetrafluoroborate form of 4-HMBD evoked tumors of the subcutis and skin, while the administration of A resulted in no detectable tumorigenic effect. Analytical chemistry investigations also confirmed the presence of A up to 0.2% in the mushroom samples. Furthermore, 4-HMBD was identified in acidic and neutral aqueous extracts of Agaricus bisporus in amounts up to 0.6 ppm. Biochemical studies carried out here also indicated that 4-HMBD is generated from A by an enzyme system present in Agaricus bisporus acting in situ. Finally, if one of the breakdown pathways of A in the mouse gut proceeds via 4-HMBD, this highly labile product is likely to decompose to 4-hydroxy-benzyl alcohol (4-HBA). In this laboratory small amounts of 4-HBA in mouse gut were detected after feeding A. (no Refs)

11. INDUCTION OF RENAL CELL TUMORS IN F344 RATS BY ORAL ADMINISTRATION OF POTASSIUM BROMATE, A FOOD ADDITIVE.

Kurokawa Y, Hayashi Y, Maekawa A, Takahashi M, Kokubo T
Div. Pathology, Natl. Inst. Hygienic Sciences, Kamiyoga 1-18-1, Setagaya-ku, Tokyo 158, Japan
Gan; 73(2):335-338 1982

Carcinogenicity of potassium bromate (K bromate) was studied in F344 rats administered K bromate (500 or 250 ppm) po in distilled water for about 2 yr. In males treated with 500 ppm, the concentration was reduced to 400 ppm at wk 60 due to inhibition of body wt increase. Control rats were given distilled water only, and each dose group consisted of 53 male and 53 female rats. Treatment with K bromate was terminated wk 110, and all surviving animals were sacrificed at wk 111 and examined. Most kidneys were sectioned in 10-15 slices 4 micrometer thick, and examined with hematoxylin and eosin stain. Mean survival time (88.1 +-18.2 wk) was shortest in males given 500 ppm K bromate; survival times of other groups were between 101-104 wk. Survival ratios at wk 104 were relatively high in the female rats treated with the K bromate. The first renal cell tumor was observed in a male rat which died wk 14; mean induction time for renal cell tumors ranged from 90 wk in males treated with 500 ppm K bromate to 111 wk in control male rats. Incidences of renal cell tumors in male rats were 90.4% (500 ppm), 58.5% (250 ppm), and 5.7% (controls); those in female rats were 69.2% (500 ppm), 50.0% (250 mm), and 0% (controls). The values for the test groups were all significantly different than those for controls. Microscopically, the renal cell tumors were adenomas or adenocarcinomas. More than 80% of the tumors were classified adenocarcinomas. Often, both adenomas and adenocarcinomas were observed in the same rat. The results indicate that po K bromate induces renal cell tumors in both male and female F344 rats and that the incidences were dose-dependent. (14 Refs)

12. MUTAGENICITY OF OXYGEN FREE RADICALS.

Moody CS, Hassan HM
(c/o Hassan) Dept. Microbiology and Food Science, North Carolina State Univ., Raleigh, NC, 27650
Proc Natl Acad Sci USA; 79(9):2855-2859 1982

Paraquat (1,1'-dimethyl-4,4'-bipyridinium dichloride) was used as an intracellular generator of oxygen free radicals and was found to be highly mutagenic for Salmonella typhimurium. It caused both base-pair substitution and frameshift mutations. Paraquat was much more toxic and mutagenic in a simple nutritionally restricted medium than in a rich complex medium. The mutagenicity of paraquat was dependent upon the presence of a supply of both electrons and oxygen. Cells containing high levels of superoxide dismutase (superoxide:superoxide oxidoreductase EC 1.15.1.1) were more resistant to the toxicity and the mutagenicity of paraquat than were cells containing normal levels of this enzyme. The mutagenicity of paraquat thus appears to be due to its ability to exacerbate the intracellular production of superoxide radicals. (Author abstract) (41 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

13. ZINC DEFICIENCY, ALCOHOL, AND A RETINOID: ASSOCIATION WITH ESOPHAGEAL CANCER IN RATS.

Gabrial GN, Schrager TF, Newberne PM
(c/o Newberne) Animal Pathology Lab., Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA, 02139
JNCI; 68(5):785-789 1982

Groups of Charles River Sprague-Dawley male rats were given intragastric intubations of methylbenzylnitrosamine after receiving one of the following diets for 4 weeks: control, zinc-deficient, or zinc-deficient diet plus 4% ethanol in the drinking water. One zinc-deficient group was zinc repleted after the dosing period; the other group, zinc-deficient plus 4% ethanol, received 13-cis-retinoic acid (13-cis-RA) after the dosing period. Zinc deficiency significantly enhanced esophageal tumor incidence; the addition of ethanol further enhanced tumor incidence. Zinc repletion reduced tumor incidence, whereas the addition of 13-cis-RA enhanced tumor incidence. (Author abstract) (30 Refs)

14. EFFECT OF MOLYBDENUM ON N-NITROSOSARCOSINE ETHYL ESTER-INDUCED CARCINOGENESIS IN RATS (MEETING ABSTRACT)

Wei HJ, Luo XM, Sproat HF, Yang SP
Department of Chemical Etiology and Carcinogenesis, Cancer Institute, Beijing, China
Fed Proc; 41(3):280 1982

Male weanling rats of the Sprague-Dawley strain were given ad libitum for 19 or 30 weeks a nutritionally adequate diet and deionized drinking water (Groups 1 and 4) or the same diet and 100 ppm W from Na2WO4 (Groups 2 and 5) or 20 ppm Mo (Groups 3 and 6) or 2 ppm Mo (Group 7) from Na2Mo0(4) added to the drinking water. As determined by a catalytic polargraphic procedure, the diet contained 0.026 ppm Mo and no detectable Mo in the drinking water. Start from the third week, all the animals in Groups 4-7 were administered via gastric intubation 0.7 g sarcosine ethyl ester and 0.3 g NaN0(2) in 10 ml 0.01 N HCl per kg of body weight twice weekly for 8 consecutive weeks. N-nitrososarcosine ethyl ester (NSEE) is believed to be formed readily under the above condition. At the end of 19 weeks, 80% of the animals in Group 4 developed esophageal and forestomach cancer. This incidence was identical to that of Group 5 and significantly higher than that of Group 6 or Group 7. Similar results were obtained at the end of 30 weeks. No carcinomas were observed in Groups 1-3. (no Refs)

15. ORAL MUCOSAL CARCINOGENESIS IN HAMSTERS: INHIBITION BY VITAMIN E.

Shklar G

Dept. Oral Medicine and Oral Pathology, Harvard Sch. Dental Medicine, 188 Longwood Ave., Boston, MA, 02115

JNCI; 68(5):791-797 1982

Eighty young adult male and female Syrian golden hamsters (Mesocricetus auratus) were divided into four equal experimental groups. In Group 1 animals the left buccal punch was painted 3x weekly with a 0.5% solution of 7,12-dimethylbenz[a]anthracene (DMBA) in heavy mineral oil. In Group 2 animal the left buccal pouch was similarly painted with DMBA, but the animals also received orally 10 mg vitamin E (dl-alpha-tocopherol) in peanut oil twice weekly on alternate days with the DMBA painting. Group 1 animals received a similar amount of peanut oil vehicle, Group 3 animals received only vitamin E (vitamin E controls) in peanut oil, and Group 4 animals served as untreated controls receiving only peanut oil. Four animals in each group (2 males and 2 females) were killed at 8, 10, 12, 14, and 16 wk. Buccal pouches were photographed and excised, and tumors were noted and measured in the left buccal pouches. In Group 2 animals receiving vitamin E, tumor formation was significantly delayed, so that by 14-16 wk there were fewer tumors and their av size was smaller than that of tumors in Group 1 animals that were painted with DMBA but received no vitamin E supplement. In Group 2 there was also less invasion of underlying tissues and less surface necrosis. (Author abstract) (30 Refs)

16. D-ALPHA TOCOPHEROL SUCCINATE (VIT E-S) AND HUMAN NEUROBLASTOMA (MEETING ABSTRACT)

Helson L, Verma M

Memorial Sloan-Kettering Cancer Center, New York, NY, 10021

Proc Am Soc Clin Oncol; 1:C-75 1982

With respect to cancer, Vitamin E is generally thought of as an antioxidant whose major activity is to reduce free radicals associated with carcinogenic activity. Based upon reports of animals tumors sensitivity to Vit E-S by K Prasad we tested three vitamin preparation's antitumor activity on an uncloned human neuroblastoma cell line SK-N-Dz. D-alpha tocopherol in Emulphor EL-620 were no more active than their vehicles. Ethyl alcohol used to solubilize Vit E-S in concentrations as high as 2% did not affect cultured tumor cells. Using an in vitro microtiter assay technique or cells grown in 75 cm2 flasks, Vit E-S in alcohol was added to culture media in final concentrations of 20-200 ug/ml with 0.2%-2.0% alcohol respectively. Within 48 hours significant antitumor activity, ie, lysis, cell growth inhibition and inhibition of 3H-thymidine incorporation occurred at concentrations greater than 50 ug/ml. Cells exposed to Vit E-S greater than 100 ug/ml for 24 hours were irreversibly damaged while no antitumor effects were noted below 25 ug/ml. 3H-uridine incorporation into RNA was not effected. At sublethal concentrations causing less than 100% kill, cell growth was temporarily perturbed with rapid regrowth of remaining cells. After single intravenous (IV) injection of D-alpha (methyl-3H) tocopherol its distribution in nude mice bearing the neuroblastoma SK-N-Dz was; lung greater than liver greater than spleen greater than kidney greater than brain greater than muscle and tumor. The IP route distribution was spleen greater than eye greater than lung greater than kidney, liver and brain. (1 Ref)

17. PROTECTIVE EFFECT OF NICOTINAMIDE ON BRACKEN FERN INDUCED CARCINOGENICITY IN RATS.

Pamukcu AM, Milli U, Bryan GT

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Nutr Cancer; 3(2):86-93 1981

Albino noninbred weanling male and female rats were fed a basic grain diet (Group 1), a basic diet supplemented with 0.5% nicotinamide (Group 2), a basic diet containing 33% bracken fern (BF) (Group 3), or a basic diet supplemented with 33% BF and 0.5% nicotinamide (Group 4) for 58 wk. Dietary nicotinamide decreased the BF-induced incidence of both intestinal and bladder tumorigenesis by about 40%. The inhibitory effect of nicotinamide on the BF-induced intestinal and bladder tumors was significant at p less than 0.05 and p less than 0.01, respectively. (Author abstract) (39 Refs)

18. THE EFFECTS OF ASCORBIC ACID AND SORBIC ACID ON N-NITROSAMINE FORMATION IN A HETEROGENEOUS MODEL SYSTEM.

Massey RC, Forsythe L, McWeeny DJ

Food Lab., Ministry Agriculture, Fisheries and Food, Haldin House, Queen St., Norwich NR2 4SX, England

J Sci Food Agric; 33(3):294-298 1982

The effects of ascorbic acid (AA) and sorbic acid (SA) as nitrite scavengers in a meat-simulating system of 20% non-aqueous solvent, 54% aqueous buffer, 18% protein, and 8% sodium carboxymethylcellulose were determined. Nitrosamines were analyzed by gas chromatography. Reactions were carried out at pH 5.25 for 50 hr at 37 C to ensure that detectable levels of nitrosamines were formed. Addition of decane to the reaction mixture increased N-nitrosodibutylamine (NDBA) formation four-fold compared to the mixture with glycerol tributyrate (GTB). With GTB, there was a four-fold increase in N-nitrosopyrrolidine (NPYR) formation, which was ascribed to greater pyrrolidine partitioning in this more polar non-aqueous phase. In the absence of decane both NDBA and NPYR formation were reduced by addition of AA, but with decane present AA had no major effect after 50 hr. When AA was replaced with SA, NPYR production was reduced 50% in the control aqueous system, as with AA, but a 50% reduction was also observed in the GTB system, evidently because of greater SA solubility in the non-aqueous phase. Situations that enhance amine solubility in the non-aqueous phase also enhance nitrosation, but the effect can be reduced by the use of an additive which is soluble in that phase. (14 Refs)

19. SEPARATION OF NATURALLY OCCURRING FORMS OF VITAMIN A BY REVERSE PHASE HIGH PRESSURE LIQUID CHROMATOGRAPHY IN RATS BEARING TUMORS (MEETING ABSTRACT)

Douglass WC, Zollinger RE, Morre DM

Purdue University, W. Lafayette, IN, 47907

Fed Proc; 41(3):387 1982

Numerous studies have demonstrated that vitamin A and its derivatives have a chemopreventative effect on experimental tumorigenesis. Until recently, the study of vitamin A metabolism in tumors has been hampered by lack of methods to separate vitamin A forms. In this study, we used reverse phase high pressure liquid chromatography (HPLC) to separate the naturally occurring forms of vitamin A in livers and tumors of

male weanling rats fed basal AIN-76A diets supplemented with zero, adequate (4,000 IU/kg diet) or excess (100X adequate) amounts of vitamin A as retinyl acetate. After two weeks on the diets, the rats were implanted with a transplantable hepatocellular carcinoma. Tissue samples were lyophilized to dryness and the residue extracted with chloroform-methanol and portions chromatographed at ambient temperature on a C18 uBondapak column using acetonitrile-1% ammonium acetate in water as the mobile phase and a flow rate of 1.8 ml/min. The amount of vitamin A in the tissues closely paralleled that in the diet. Chromatographic profiles of all tissues assayed generally showed small amounts of retinoic acid and retinyl acetate, moderate amounts of retinol and the long chain retinyl esters in greatest abundance. Reverse phase HPLC is especially useful for tumor tissues where all forms of vitamin A are reduced in amount compared to liver. (no Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

20. DIETARY MODIFICATION OF METABOLISM AND TOXICITY OF CHEMICAL SUBSTANCES - WITH SPECIAL REFERENCE TO CARBOHYDRATE.

Nakajima T, Koyama Y, Sato A
(c/o Sato) Dept. Hygiene, Shinshu Univ. Sch. Medicine, Asahi, Matsumoto, Japan
Biochem Pharmacol; 31(6):1005-1011 1982

Rats were fed various test diets only on the day before sacrifice or every day for 3 wk prior to sacrifice in order to assess the effects of protein (casein), fat (a mixture of olive and corn oils) and carbohydrate (sucrose) on the liver mixed-function oxidase activity. The activity was determined by measuring metabolic rates of eight volatile hydrocarbons, ie, benzene, toluene, styrene, chloroform, carbon tetrachloride, 1,2-dichloroethane, 1,1-dichloroethylene, and trichloroethylene. Contrary to the general belief, it was found that carbohydrate, not protein or fat, regulates the metabolism of these hydrocarbons; a diet which was deficient in carbohydrate remarkably enhanced the metabolism irrespective of protein and fat contents in the diet. This conclusion was confirmed by employing two types of diet, one in which the carbohydrate was replaced by an isocaloric amount of protein or fat (thus keeping total calories of each diet constant) and the other in which the carbohydrate content was varied with protein and fat contents fixed (total calories of each diet differed from others according to the carbohydrate content). In accordance with this, dietary carbohydrate intake also exerted a remarkable influence on the hepatotoxocity of carbon tetrachloride which needs to be metabolically activated to become cytotoxic; the smaller the intake, the more severe the liver injury. (Author abstract) (28 Refs)

21. MALNUTRITION, LIVER DAMAGE, AND CANCER.

Grasso P
British Petroleum Group Occupational Health Centre, Chertsey Road, Sunbury-on-Thames, Middlesex TW16 7LN, England
Nutr Cancer; 3(2):103-108 1981

There is no clear indication that malnutrition, per se, is a principal cause of cancer in man, but the prevalence of liver cancer in areas where malnutrition exists supports this hypothesis. Liver damage and liver cancer have been induced in laboratory rats by diets consisting of peanut meal and proteins deficient in some essential amino acids. However, liver damage, but not cancer, was produced when the diets contained no peanut meal but consisted of a mixture of amino acids deficient in methionine and cysteine, so that it is possible that aflatoxin, a contaminant of peanut meal, may have been responsible for the malignancies seen in the earlier experiments. Liver cancer develops in a high proportion of mice allowed to feed ad libitum or given a diet containing a high proportion of fat (groundnut oil) or protein (casein). Dietary restriction reduced the incidences of this cancer. This finding lends some support to current thinking that diet may be a factor in the development of cancer in man. (Author abstract) (23 Refs)

22. EARLY PROLIFERATIVE CHANGES IN GASTROINTESTINAL NEOPLASIA.

Deschner EE
Lab., Digestive Tract Carcinogenesis, Memorial Sloan Cancer Center, 1275 York Ave., New York, NY, 10021
Am J Gastroenterol; 77(4):207-211 1982

The proliferative behavior of mucosal epithelium is reviewed in relation to the development of gastrointestinal cancer. An enlarged proliferative compartment has been recognized among individuals with a previous history of colon cancer, those with an isolated polyp, asymptomatic relatives of familial polyposis patients, those with ulcerative colitis, and even some members of the general population. Mucosa of patients with a previous history of colon cancer may show both extension of the proliferative compartment to the luminal surface (Stage I defect) and a shift in the major zone of DNA synthesis from the lower third of the gland to the middle and upper portion of the crypt (Stage II defect). Studies of 1,2-dimethylhydrazine-induced colon carcinogenesis in mice indicate that Stage II abnormalities are predictive of the development of cancer. Distribution of DNA synthesizing cells among patients belonging to several high risk groups is presented. Only 30% of the patients showed a completely normal proliferative pattern. Changes in proliferative patterns over time are described for two asymptomatic members of polyposis coli families. Histologically normal mucosa from patients with a previous history of neoplasia may show crypts with extremely elevated labeling indices. The importance of such crypts is discussed. Correlation of an abnormal proliferative pattern with the type of tumor that will emerge is described. The impact of a diet rich in animal and dairy fats is considered. (16 Refs)

23. GLUTATHIONE S-TRANSFERASE ACTIVITY: ENHANCEMENT BY COMPOUNDS INHIBITING CHEMICAL CARCINOGENESIS AND BY DIETARY CONSTITUENTS.

Sparnins VL, Venegas PL, Wattenberg LW
Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN, 55455
JNCI; 68(3):493-496 1982

Benzyl isothiocyanate, beta-naphthoflavone, coumarin, alpha-angelicalactone, disulfiram, indole-3-carbinol and indole-3-acetonitrile induced increased (GSH) glutathione S-transferase activity in the liver and small intestine in female ICR/Ha mice. All seven compounds are inhibitors of chemical carcinogenesis. In additional work, several dietary constituents increased GSH S-transferase activity. Consumption of diets containing dried powdered preparations of brussel sprouts, cabbage, coffee beans, or tea leaves resulted in increased GSH S-transferase activity. Mice fed an unrefined diet (Purina Rat Chow) had a higher GSH S-transferase activity than those fed a semipurified diet. The results of the present study indicated

that the composition of the diet can alter the activity of an important enzyme system having the capacity to detoxify chemical carcinogens. (Author abstract) (28 Refs)

24. MEAT AND FAT CONSUMPTION AND CANCER MORTALITY: A STUDY OF STRICT RELIGIOUS ORDERS IN BRITAIN.

Khulen LJ

Cancer Res. Campaign, Cancer Epidemiology Res. Group, Univ. Oxford, Radcliffe Infirmary, Oxford, England

Lancet; 1(8278):946-949 1982

The close correlations between mortality from breast and colorectal cancers and the per-caput consumption of meat or fat in different countries suggest that these dietary items may be involved in the etiology of these neoplasms. To investigate further these possible relations a study has been conducted of cancer mortality between 1911 and 1978 in two groups of enclosed religious orders for women: one of 1,769 nuns who eat no meat and one of 1,044 nuns who eat little meat. Mortality from breast and colorectal cancer was not significantly lower in either group than in the general population. It is difficult to evaluate the small reduction in breast cancer mortality in the no-meat group because of the wide confidence limits. An unexpected finding was an excess mortality from esophageal cancer in both groups. These findings concern women who reduced their meat consumption in adult life: it is suggested that they are compatible with the evidence put forward to support a role for fat or meat intake in causation of breast cancer if dietary influences in pre-adult life are important. The findings on colorectal cancer point to the etiological importance in this disease of aspects of diet other than fat or meat intake. (Author abstract) (34 Refs)

25. TRENDS IN DIET AND BREAST CANCER MORTALITY IN ENGLAND AND WALES 1928-1977.

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Nutr Cancer; 3(2):75-80 1981

Trends in age-adjusted breast cancer mortality and consumption of meat, fat, sugar, cereal, and fruit and vegetables were studied for England and Wales over the 50-yr period from 1928 to 1977. At the onset of World War II, there was a marked reduction in both breast cancer mortality and intake of sugar, meat and fat, and an increased consumption of cereals and vegetables. Consumption of these foodstuffs returned to pre-war levels by 1954, but breast cancer mortality did not return to pre-war levels until some 15 yr later. The association between the various dietary components and subsequent breast cancer mortality was determined for various lag intervals. Significant correlations were found for cereal, fat, sugar and meat consumption, the correlation being maximal for a diet-breast cancer death lag interval of 12 yr. These findings add weight to the hypothesis that breast cancer development is related to a diet rich in meat, fat and sugar, and that some protection against cancer may be afforded by a reduction in these dietary components and an increase in cereal consumption. (Author abstract) (20 Refs)

26. HIGH RISK GROUP FOR PROSTATIC CANCERS BY MATCHED PAIR ANALYSIS.

Mishina T, Watanabe H, Araki H, Miyakoda K, Fujiwara T, Kobayashi T, Maegawa M

Dept. Urology, Kyoto Prefecture Univ. Medicine, Kawaramachi-Hirokoji, Kyoto, Japan

Nippon Hinyokika Gakkai Zasshi; 72(10):1256-1279 1981

In order to find the high risk group for prostatic cancer, a questionnaire was designed, and 100 patients (47-86 yr old, av age 68.7) with prostatic cancers and 100 matching people as control group were interviewed. The results were analyzed with matched pain analysis. A peak was seen in the 60-70 yr old group of the 100 patients. No statistical differences were seen between the groups on the following questions: education, income, religious preference, present marital status, body characteristics such as balding, white hair, beard, etc, and familial history of illness. Significant differences were found on a number of other questions. The high risk was found in those who are not in management, had no military experiences, were married for more than 40 yr, married at an early age, had experienced ejaculation before age 14, had frequent intercourse below age 50 but less frequent over age 51, had past history of illness in urinary system and surgical experiences, prefered spicy and salty food, ate fish and vegetable less often, and drank alcohol and smoked. These findings suggest that the high risk group for prostatic cancer can be found in those people who have specific pattern in sex life and eating habits. (63 Refs) (In JPN)

27. INHIBITORY EFFECT OF DIFFERENT DIETARY COMPONENTS ON NITROSAMINE FORMATION AND NITRITE LEVELS (MEETING ABSTRACT)

Higgy NA, El-Aaser AA, El-Merzabani MM

Department of Cell Biology, Cairo Cancer Institute, Cairo University, Cairo, Egypt

Fed Proc; 41(3):356 1982

Different vegetables, fruit juices, bread, or yogurt were homogenized and incubated with piperazine and nitrite at pH 4.0 in acetate-HCl buffer. Nitrosamine formation was significantly inhibited as compared to controls. However, with higher levels of nitrite, nitrosamine levels increased. When these dietary homogenates were incubated with nitrite, nitrite levels decreased gradually and then disappeared completely from incubation mixtures. Vegetables like watercress, radishes, lettuce and shikori had high levels of endogenous nitrate, but no nitrite. When these were incubated with nitrite, nitrite levels gradually decreased; then a marked increase occurred after 18 hours of incubation and was attributed to the reduction of the endogenous nitrate. Vegetables and different foodstuffs may have a protective action by inhibiting or interfering with nitrosamine formation. This may be attributed to ascorbic acid, tannins, alpha-tocopherol or other inhibitors of nitrosation, or substances competing with the nitrosatable amine, such as urea and primary amines. This effect may also be due to the disappearance or uptake of nitrite. (no Refs)

28. EFFECT OF DIETARY PROTEIN ON INDUCTION OF 7,12-DIMETHYLBENZ(A)ANTHRACENE MUTAGENESIS BY RAT LIVER MICROSOMES IN THE SALMONELLA/ MICROSOME TEST SYSTEM (MEETING ABSTRACT)

Singletary KW, Martin SE, Milner JA
Department of Food Science, University of Illinois, Urbana, IL, 61801
Fed Proc; 41(3):356 1982

Studies have shown that a high protein diet decreases the incidence of 7,12-dimethylbenz(a)anthracene (DMBA)-induced tumors in rats. The present studies were designed to evaluate the influence of dietary protein on the ability of rat liver microsomes to mediate DMBA-induced mutagenesis. Female rats (150g) were fed isocaloric diets containing 7.5, 15.0 or 45.0% protein as casein for 2 weeks. All rats were treated with Aroclor (100mg/kg) 5 days before sacrifice. Liver homogenates (S-9) were prepared and evaluated for their influence on the mutagenicity of DMBA (20 g/plate) with Salmonella typhimurium TA98. S-9 fractions from rats fed the 7.5% protein diet produced approximately 100% more DMBA-induced revertants per mg protein than S-9 fractions from rats fed the 15 or 45% protein diets. Pair feeding studies revealed food intake did not account for these differences. The duration of feeding was also found to influence the S-9 activation effects of dietary protein. These results suggest that the Salmonella/microsome mutagenesis assay has potential use as a relatively simple and rapid system to evaluate the influence of dietary protein source and concentration as factors modifying carcinogenesis. (no Refs)

29. PRECURSOR LESIONS OF OESOPHAGEAL CANCER IN HIGH-RISK POPULATIONS IN IRAN AND CHINA.

Munoz N, Crespi M, Grassi A, Qing WG, Qiong S, Cai LZ
Analytical Epidemiology Program, Div. Epidemiology and Biostatistics, International Agency for Res. on Cancer, 150 Cours Albert-Thomas 69372, Lyon Cedex 2, France
Lancet; 1(8277):876-879 1982

To determine the prevalence of precursor lesions of esophageal cancer in Linxian, People's Republic of China, which has an extremely high incidence of esophageal cancer, 527 subjects were interviewed by a questionnaire about exposure to suspected risk factors. They also underwent esophagoscopy, cytology, and biopsy. 84% had a chronic esophagitis which had similar endoscopic and histological characteristics to that reported in Iran. The esophagitis was accompanied by atrophy of the epithelium in 10% of these subjects and by dysplasia in 8%. Reexamination of 20 selected subjects 1 year later showed progression of the esophagitis to atrophy and dysplasia in 4 individuals and to cancer in 4 others. The surveys in Iran and China suggest that the natural history of esophageal cancer starts with an esophagitis which, in a few individuals, progresses to atrophy and dysplasia of the epithelium and finally to cancer. Thermal injury resulting from the drinking of very hot beverages, physical injury caused by ingesting very coarse food, and deficiencies of riboflavin, vitamin A, and zinc are proposed as risk factors for these precursor lesions. (Author abstract) (17 Refs)

GENERAL REVIEWS AND OTHER STUDIES

30. NATURALLY OCCURRING FOOD TOXICANTS: PHENOLIC SUBSTANCES OF PLANT ORIGIN COMMON IN FOODS.

Singleton VL
Dept. Viticulture and Enology, Univ. California, Davis, CA
Adv Food Res; 27:149-242 1981

The common phenolic substances of the plant kingdom are reviewed as real or potentially toxic or physiologically active substances in human and animal diets. Origins and types of plant phenols, evolutionary considerations, examples of plant phenols with actual or potential significance (toxic or beneficial) in animal consumption, mechanisms of toxicity by phenols, and risk assessment are discussed. (479 Refs)

31. THE SAME EVIDENCE - WHY DIFFERENT CONCLUSIONS?

Walker AR
South African Inst. for Medical Res., P.O. Box 1038, Johannesburg 2000, South Africa
S Afr J Sci; 77(12):534-537 1981

Controversy exists as to the etiology of cancer and coronary heart disease and roles of dietary fiber and sugar in health and disease. The same body of evidence may be analyzed by different researchers with a different conclusion by each. As an example, out of all deaths from lung cancer in the United States, 20% occur in nonsmokers. A large proportion of cancer related deaths must occur independent of smoking and life style. One group contends that occupational carcinogens are vastly underestimated while another contends that this view is not mutually exclusive. The role of diet in coronary heart disease is also discussed in the light of recent epidemiologic statistics. (65 Refs)

32. MUTAGENS, CARCINOGENS, AND TUMOR PROMOTERS IN OUR DAILY FOOD.

Sugimura T
Natl. Cancer Res. Inst., Tsukiji, Chuo-ku, Tokyo, Japan
Cancer; 49(10):1970-1984 1982

Identification of mutagens, carcinogens, tumor promoters in human food is reviewed. Studies of the food additive 2-(2-furyl)-3-(5-nitrofuryl)acrylamide, which is mutagenic in the Ames test and produced tumors in mice, indicated that if a compound in food shows mutagenicity, its carcinogenicity should be considered and that data obtained on mutagenicity prior to 1960 may be invalid. Improvement and adoption of the Ames test for screening for carcinogens in food are discussed. Isolation and identification of new heterocyclic amines from pyrolysates of amino acids, proteins, and proteinous foods are described. Mutagens and carcinogens in edible plants, vegetables, fruits, alcoholic spirits, spices, and beverages are described. Tumor promoters are discussed in relation to life style. Recommendations are proposed for cancer prevention. (159 Refs)

33. ETIOLOGY OF MOUTH AND LIP TUMORS.

Zabezhinskii MA, Godorozha PD
N. N. Petrov Res. Inst. Oncology, Leningrad, USSR
Vopr Onkol; 28(2):105-113 1982

Literature data on the etiology of mouth and lip cancers are reviewed. Pathogenesis of mouth neoplasms is associated with the exposure to various chemical, physical, and biological carcinogenic factors alone or in combination with the modifying factors. The increased incidence of mouth cancer can be caused by smoking, tobacco chewing, exposure to polycyclic aromatic hydrocarbons, alcohol consumption, and exposure to pesticides. Physical carcinogens include UV, x rays, and sunlight. Experimental data indicate that herpes virus and polyoma virus can induce adenomas and adenocarcinomas of mouth mucosa. Poor hygiene of the mouth can increase the risk of development of mouth cancer. Chronic irritation of the mouth mucosa was shown to enhance penetration of carcinogens into mucosa and to stimulate cell proliferation. Bacterial flora of the oral cavity was shown to contribute to the metabolism of chemical carcinogens. Deficiency of vitamins A and B led to development of precancerous conditions of the mouth mucosa. (74 Refs) (In RUS)

34. FUSARIUM MYCOTOXINS AND THE EFFECTS OF HIGH-FAT DIETS.

Schoental R
Dept. Pathology, Royal Veterinary Coll., Univ. London, Royal College St., London NW1 0TU, England
Nutr Cancer; 3(2):57-62 1981

Some of the inconsistencies encountered in epidemiological and experimental studies on the role of various types of fats in the etiology of cardiovascular disorders and certain tumors (of the breast, endometrium, and colon, among others) may be related to the presence of adventitious contaminants in the fats. Among the contaminants, mycotoxins, especially those produced in agricultural products by the Fusarium field-microfungi (the irritant trichothecenes, the estrogenic zearalenone, and other resorcylic acid lactones), warrant particular attention. Their production is favored by the wet, cool weather prevailing in areas notable for high incidences of the aforementioned disorders. The recent development of sensitive radioimmunoassays for the detection and estimation of some of these mycotoxins should facilitate the testing of the above hypothesis. (Author abstract) (39 Refs)

35. MUTAGENS IN HEATED FOODS.

Pariza MW
Food Res. Inst., Dept. Food Microbiology and Toxicology, Univ. Wisconsin, Madison, WI, 53706
Food Technol; 36(3):53-56 1982

Mutagens in heat-processed foods are discussed with respect to effects of time and temperature on mutagen formation, chemical inhibition of mutagenesis and carcinogenesis, and safety considerations. Formation of carcinogenic polycyclic aromatic hydrocarbons and tryptophan on broiled meats is dependent on relatively high temperature; however, mutagenic activity recently has been found in a variety of foods processed under more moderate conditions. Temperature/time evaluations in ground beef during frying have demonstrated that mutagen formation is temperature dependent: Mutagenic activity readily developed at frying temperatures of 191 C or 210 C, whereas only low levels of mutagenic activity was observed at all time intervals during frying at 143 C. A number of chemicals, including beta-naphthoflavone, butylated hydroxytoluene, butylated hydroxyanisole, and polycyclic aromatic hydrocarbons, inhibit cancer in animals when fed before or during carcinogen administration, apparently via enzyme induction. Epidemiologic studies indicate a correlation between increased cancer incidence at some sites and diets high in charred and/or heavily smoked foods. Experimental evidence, however, does not suggest that mutagens in the diet play an important role in this correlation. (26 Refs)

36. OCCURRENCE OF MUTAGENIC PRODUCTS IN BROWNING MODEL SYSTEMS.

Shibamoto T
Dept. Environmental Toxicology, Univ. California, Davis, CA, 95616
Food Technol; 36(3):59-62 1982

Studies suggest that some browning products may play a role in the mutagenicity of tars obtained from cooked foods. Browning model systems consisting of simple amines and carbonyls have been used to study the nature of cooked foods and can play an important role in the investigation of chemical mutagenicity and carcinogenicity. Results obtained from maltol/NH3 and rhamnose/NH3/H2S model systems are presented. Major components of browning reaction mixtures are nitrogen-containing compounds such as pyrazines and imidazoles. Some of these compounds are mutagenic in the Ames test, Saccharomyces cervisiae, or Chinese hamster ovary cells, indicating the necessity of further mutagenicity testing. (35 Refs)

37. MUTAGENESIS VS CARCINOGENESIS.

Taylor SL
Food Res. Inst. Dept. Food Microbiology and Toxicology, Univ. Wisconsin, Madison, WI, 53706
Food Technol; 36(3):65-68,98,100,102-103,127 1982

Factors influencing the predictive value of mutagenicity tests for detecting carcinogens include the nature of the mutagenesis system; the population, identity, and nature of the chemicals being tested; the chemical classes of the compounds being tested; the reliability of the data; and the nature of any activating system that may be involved. Short-term mutagenicity tests may be useful for identifying food components that warrant further toxicologic evaluation and for studying the mechanism of carcinogenesis. They may eventually replace long-term carcinogenicity tests in animals; data generated by the present short-term tests, however, must be interpreted with caution. (65 Refs)

38. NITROSAMINES.

Aigner K
Pneumonologische Abteilung, Krankenhaus der Elisabethinen, Fadingerstr. 1, A-4020 Linz-Donau, Austria
Wien Med Wochenschr; 132(71,Suppl):75-79 1982

Health hazards of nitrosamines (NA) are reviewed. NA are formed by the reaction of amines or amides with nitrite or nitrous gases. NA are found in foods, cosmetics, technical oils, pesticides, tobacco, and drugs. Elevated levels of NA have been found in patients with some diseases, including gastric carcinoma. Animal studies have confirmed the carcinogenicity of NA. Further research is required to reduce intake and formation of NA in humans. (17 Refs) (In GER)

39. MAMMARY CANCER: HIGH RISK FACTORS FOR ITS DEVELOPMENT.

Torres Lobaton A, Diaz Perches R, Marquez GA, Miranda Hernandez H
Unidad de Oncologia, Hospital General de Mexico, S.S.A., Mexico, DF, Mexico
Rev Med Hosp Gen Mex; 44(8):281-287 1981

High risk factors in development of mammary cancer (MC) were discussed, and the following aspects were included age, height, wt, heredity, dietary factors, personal history of MC, benign mammary pathology, exposure to ionizing irradiation, menstruation, menopause, parity, and hormones. Intake of estrogens under any form especially after menopause was also considered. (50 Refs) (In SPA)

40. EPIDEMIOLOGY AND EVOLUTION OF GASTRIC CANCER.

Li Voti P
Via Principe di Villafranca, 99, 90141 Palermo, Italy
Minerva Chir; 36(23/24):1709-1712 1981

Between 1975-1976, gastric cancer (GC) mortality was 28.2/100,000 inhabitants in Italy; 2.90% of the total mortality, 14.2% of the total mortality because of tumors, and 42.93% mortality because of tumors of the digestive tract. Incidence of the GC was highest in the province of Emilia-Romagna (199) and Toscana (169) in the central part of the country, and lowest in Sardegna (27) and Puglie (29) in the south. GC frequency is decreasing in Italy by 16.5%, after a peak was reached in 1960-1969. The relationship between GC and nutrition, age, sex, and genetic factors (blood group) were discussed. Different gastric pathologies like atrophic gastritis, giant hypertrophic gastritis (Menetriere's disease), gastric polyposis, and previous gastric resection increase the probability of GC. Canceration of gastric epithelium is very slow. Span of time between appearance of early cancers and appearance of symptoms is slow too, time can vary, but is basically unknown. Survival for patients with GC who were not cured was 2-147 mo after the symptoms had started or 1-145 after the diagnosis. (12 Refs) (In ITA)

41. RETINOIDS, URINARY BLADDER CARCINOGENESIS, AND CHEMOPREVENTION: A REVIEW AND SYNTHESIS.

Lower GM, Kanarek MS
Dept. Veterinary Science, Coll. Agricultural and Life Sciences, Univ. Wisconsin, 1655 Linden Drive, Madison, WI, 53706
Nutr Cancer; 3(2):109-115 1981

An analysis of the effects of natural (vitamin A) and synthetic retinoids on experimental carcinogenesis of the urinary bladder indicates that inhibitory activity is carcinogen- or carcinogen-class-specific. These findings suggest that there may be more than one pathway for the neo-differentiation of urothelial cells to frank carcinoma and that these pathways may be related to both carcinogen class and dietary vitamin A status. (Author abstract) (54 Refs)

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As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

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In DAN

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Larsen B
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Neiman IM
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ENVIRONMENTAL AND OCCUPATIONAL FACTORS IN CARCINOGENESIS

21. POLYCHLORINATED BIPHENYLS (PCBS): AN AUSTRALIAN VIEWPOINT ON A GLOBAL PROBLEM.
Richardson BJ, Waid JS
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Hochhauser SJ, Stein GS, Stein JL
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Lower GM, Kanarek MS
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Cancer; 49(10):1958-1962 1982

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Weiss RA
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Symp Soc Gen Microbiol; 33:267-288 1982

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NCI, Bethesda, MD
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Cold Spring Harbor Lab., Box 100, Cold Spring Harbor, NY, 11724
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SERIES CK03 No. 82/10 OCTOBER, 1982

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STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. DETECTION OF MUTAGENIC ACTIVITY IN HUMAN URINE FOLLOWING FRIED PORK OR BACON MEALS.

Baker R, Arlauskas A, Bonin A, Angus D
Commonwealth Inst. Health, Univ. Sydney, Sydney, New South Wales 2307, Australia
Cancer Lett; 16(1):81-89 1982

The mutagenic activity of urine from nonsmoking individuals following meals of fried pork and bacon was tested in the Salmonella typhimurium revertant assay. The greatest degree of mutagenicity was observed in urine collected 2-4 hr after ingesting the fried test foods. Evidence of mutagenic activity continued for up to 24 hr. In contrast, ingestion of microwave prepared pork products failed to produce urinary mutagens. Urine mutagenicity was unrelated to histidine concentration in urine because of the requirement for S9. Beta-glucuronidase did not enhance the activity of urine mutagens either when incorporated together with urine extracts in agar overlays, when preincubated with urine extract, or with various whole urine specimens. Mutagenicity was acetone extractable from fried

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but not from microwave-prepared or raw pork, and was associated with the basic fraction rather than the acidic plus neutral fraction. Activity was best detected in strains TA1538 and TA98. Liver S9 was required to detect mutagenicity in these meat extracts. Mutagenic extracts also increased the frequency of sex-linked recessive mutations in Drosophila melanogaster. (19 Refs)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

2. A CASE-CONTROL DIETARY STUDY OF PRIMARY LIVER CANCER RISK FROM AFLATOXIN EXPOSURE.

Bulatao-Jayme J, Almero EM, Castro CA, Jardeleza TR, Salamat LA
Food and Nutrition Res. Inst., PO Box EA 467, Ermita, Manila 2801, Philippines
Int J Epidemiol; 11(2):112-119 1982

The dietary intakes of 90 confirmed primary liver cancer cases were compared against 90 age-sex matched controls. Dietary recall, obtained by the same nutritionist, was taken for each change of residence lasting 1 yr or more. The frequency and amounts of food items consumed were calculated into units of aflatoxin load per day using a Philippine table of aflatoxin values of these items. Of the total subjects' aflatoxin load, 51.2% came from cassava, 20.3% from corn, 6.8% from peanuts and 5.8% from sweet potato. The mean aflatoxin load per day of the cases as found to be 440% that of the controls. Upon grouping dietary aflatoxin loads (and alcohol intakes) into Heavy and Light, and upon comparing cases versus controls, the relative risk (RR) of developing primary liver cancer was found to be statistically significant in the following order of rank: cassava, peanuts, sweet potato, corn and alcohol. Boiled rice which had negligence aflatoxin content gave no difference in risk. Combining aflatoxin load and alcohol intake gave a synergistic and a statistically significant effect on RR with Light Aflatoxin, Light Alcohol as RR = 1. Results: Light Aflatoxin, Heavy Alcohol RR = 3.9; Heavy Aflatoxin, Light Alcohol RR = 17.5; Heavy Aflatoxin, Heavy Alcohol RR = 35.0. (Author abstract) (25 Refs)

3. CHOLANGIOCELLULAR CARCINOMAS INDUCED IN SYRIAN GOLDEN HAMSTERS ADMINISTERED AFLATOXIN B1 IN LARGE DOSES.

Moore MR, Pitot HC, Miller EC, Miller JA
(c/o Miller) McArdle Lab. Cancer Res., Univ. Wisconsin Medical Sch., 450 North Randall Ave., Madison, WI, 53706
JNCI; 68(2):271-278 1982

The hepatocarcinogenicity of aflatoxin B1 (AFB1) was compared in male Syrian golden hamsters and inbred F344 rats. AFB1 was administered by gavage 5 days/wk for 6 wk at doses of 1 and 2 mg/kg body wt/day to rats and hamsters, respectively; rats did not survive beyond 3 wk with doses of 2 mg/kg/day. After 6 wk the animals either received no further treatment or were given 0.1% phenobarbital sodium in the drinking water. ATPase-deficient foci of hepatic parenchymal cells, neoplastic nodules, and hepatocellular carcinomas were observed in liver sections from AFB1-treated rats killed at 6, 14, or 23 wk; they were not seen in sections from AFB1-treated hamsters killed at 6, 14, or 46 wk. Each of the 50 AFB1-treated rats developed hepatocellular carcinomas by 46 wk; many also had cholangiocarcinomas and mixed hepatocellular-cholangiocellular carcinomas. Hepatocellular carcinomas were found in only 2 of 49 AFB1-treated hamsters by 78 wk. At this time cholangiomas were found in 15 hamsters, and microscopic cholangiomas were seen in all of the livers. Compared to the rat, the hamster was resistant to the hepatotoxic and hepatocellular carcinogenic effects of AFB1. (Author abstract) (32 Refs)

4. BLADDER CANCER: ARTIFICIAL SWEETENERS (MEETING ABSTRACT)

Connolly JG, Chapman JA, Rider WH
No affiliation given
77th Annual Meeting of the American Urological Association, Inc., May 16-20, 1982, Kansas City, Missouri. American Urological Association 254 pp., 1982.

This is a report on a retrospective study of 249 men and 98 women with bladder cancer. Patients were obtained from the Princess Margaret, Women's College, Toronto General and Oshawa General Hospitals. Cases were matched with two controls each to examine the relationship between study factors and bladder cancer. Matching factors were sex, age within five years, and geographic area of residence. Controls were both institutional and non-institutional. Preliminary results suggested that there was little difference in the proportions of patients and controls who had used artificial sweeteners. We have now looked at the amount consumed, length of use, use of diet soft drinks, and number of diabetics. Patients and controls still appear to be similar. The effects of possible confounding variables such as cigarette smoking, coffee and tea drinking have been examined and are generally not important. However, the type of control was important; more women artificial sweetener users were institutionalized. Thus, control type may affect whether case-control differences in studies are significant. (no Refs)

5. COCARCINOGENICITY OF SACCHARIN TOWARD N-DIBUTYL-NITROSAMINE CARCINOGENICITY IN RAT LIVER AND URINARY BLADDER (MEETING ABSTRACT)

Pereira MA, Britt AL
Health Effects Research Laboratory, US Environmental Protection Agency, Cincinnati, OH
Toxicologist; 2(1):64 1982

Saccharin has been demonstrated to be a promoter of chemical carcinogenesis in the rat urinary bladder. Since other tumor promoters are also cocarcinogens, we evaluated the possibility that saccharin is also a cocarcinogen. Male Fisher 344 rats simultaneously received 0.02% dibutylnitrosamine (DBN) in their drinking water and 5% sodium saccharin in their diet for a total of 26 weeks. The liver of rats that received only DBN contained 16 hyperplastic nodules among 5 of 29 (17%) animals and 2 hepatocellular carcinomas in 1 of 29 (3%) of the animals. The simultaneous administration of sodium saccharin to the animals that received DBN, produced an enhanced hepatocarcinogenic response of 106 hyperplastic nodules in 17 of 21 (81%) animals and 81 hepatocellular carcinomas in 17 of 21 (81%) animals. The number of preneoplastic focal alterations in the liver that were induced by DBN, was also enhanced by sodium saccharin. The livers of untreated control rats an rats that received only sodium saccharin were free of preneoplastic foci, hyperplastic nodules and hepatocellular carcinomas. In the urinary bladder, treatment with DBN produced simple and nodule hyperplasia that was enhanced by simultaneous treatment with saccharin. Sodium saccharin did not result in either a preneoplastic hyperplasia or a neoplastic response in the urinary bladder of the rats. (no Refs)

6. SILICA FRAGMENTS FROM MILLET BRAN IN MUCOSA SURROUNDING OESOPHAGEAL TUMOURS IN PATIENTS IN NORTHERN CHINA.

O'Neill CH, Clarke G, Hodges G, Jordan P, Newman R, Pan QQ, Liu FS, Ge M, Chang YM, Toulson E
Imperial Cancer Res. Fund Lab., P.O. Box 123, Lincoln's Inn Fields, London WC2A 3PX, England
Lancet; 1(8283):1202-1206 1982

Millet bran is a component of the diet in the area of highest esophageal cancer incidence in northern China. Millet bran was found to contain up to 20% by wt of silica; some of this silica occurs as friable sheets or sharply-pointed fibers. These types of silica in millet bran are the most likely source of an unusual contamination with fragments of silica found in the esophageal mucosa surrounding tumors in patients in northern China. A group of mucosal samples analyzed together contained over 5,000 particles/g (100 ppm by wt), 10x as many as were found in tissue from normal controls taken at necropsy in London. The modal diameter was 10 um (1-70 um). The particles were in the body of the mucosa and were not simply a surface contaminant. Silica fragments and fibers of similar size originating from other plant species occur in the diet in the two other regions of greatest incidence of esophageal cancer, the Transkei and Iran. If such fragments enter the mucosa, they must cause some degree of trauma, and they may also be able to stimulate proliferation by providing anchorage. These findings suggest the possibility that silica particles might be involved in the etiology of esophageal cancer. (Author abstract) (16 Refs)

7. NEW CONSIDERATIONS ON HYDROGEN PEROXIDE AND RELATED SUBSTANCES AS FOOD ADDITIVES IN VIEW OF CARCINOGENICITY.

Ito R
Dept. Pharmacology, Toho Univ. Sch. Medicine, Tokyo, Japan
Paediatrician; 11(3/4):222-224 1982

The actions of potassium bromate (used as an improving agent in bread) and sodium chlorate (used as a weed killer) are under study because of the finding of hyperplasia in the duodenal mucosa of mice after long-term oral feeding with H2O2. Mutagenicity of bromate was shown in the Ames test but carcinogenicity is still under study. It has been ruled in Japan that H2O2 is safe for use in food when it is entirely decomposed. (3 Refs)

8. COMPARATIVE EFFECTS OF DIETARY ADMINISTRATION OF 2(3)-TERT-BUTYL-4-HYDROXYANISOLE AND 3,5-DI-TERT-BUTYL-4-HYDROXYTOLUENE ON SEVERAL HEPATIC ENZYME ACTIVITIES IN MICE AND RATS.

Cha YN, Heine HS
Dept. Pathobiology, Johns Hopkins Univ., 615 North Wolfe St., Baltimore, MD, 21205
Cancer Res; 42(7):2609-2615 1982

Effects of feeding mice and rats with 2(3)-tert-butyl-4-hydroxyanisole (BHA) and 3,5-di-tert-butyl-4-hydroxytoluene (BHT), have been compared to search for common effects between the two agents in two different rodent species and then applied toward better understanding of the mechanisms involved in their protective actions. Common effects could not be recognized at the microsomal mixed-function oxidase level. Contrary to expectations based on chemical structures, BHT feeding elevated the epoxide hydrolase activity to an even greater extent than that produced by BHA, especially in rats. However, enzyme activities involved in the glucuronide conjugation system are all elevated by both antioxidants in both rodent species. With BHA treatment, the levels of acid-soluble thiols were increased in both rats and mice. However, with BHT, the level was increased only in mice but not in rats. Similar trends were produced for glucose-6-phosphate dehydrogenase activity, but glutathione reductase activity was increased even for BHT-treated rats. Additionally, the glutathione S-transferase activities were also increased by both antioxidant treatments and in both rodent species. Based on these results, the elevations of epoxide hydrolase activity along with the enhanced glucuronide conjugation and glutathione oxidation-reduction conjugation system enzyme activities were common to both compounds in both rodent species. This suggests their involvement in anticarcinogenic mechanisms. Increases of these detoxification enzyme activities appeared to be all designed to accelerate the elimination of administered antioxidants but, inadvertently, conferring protective effects from xenobiotics such as carcinogens. (49 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

9. CAFFEINE AND UNSATURATED FAT DIET SIGNIFICANTLY PROMOTES DMBA-INDUCED BREAST CANCER (MEETING ABSTRACT)

Minton JP, Foecking MK, Abou-Issa H
Ohio State University College of Medicine, Columbus, OH
Joint Meeting of the Society of Surgical Oncology and Society of Head and Neck Surgeons, April 18-23, 1982, Marco Island, Florida. The Society of Surgical Oncology and the Society of Head and Neck Surgeons 265 pp., 1982.

Caffeine is an important causative agent in the development of human fibrocystic breast disease. The epithelial proliferation resulting from caffeine consumption has been shown to be reversible when caffeine abstention is carried out. The exposure of a well-established animal model to the known carcinogen dimethylbenz(a)anthracene (DMBA) is associated with the development of a hormone sensitive estrogen receptor positive breast tumor in nearly 90% of the animals. To determine the possibility of caffeine being an extraordinary promoter of breast cancer development in animals consuming vegetable fat the following study was carried out. A controlled study comparing the time to tumor development and the number of tumors and the growth rate of breast cancer in Sprague-Dawley rats which had been given 50 mg of DMBA at 6 weeks of age compared the tumor promotional effects of caffeine alone, caffeine and unsaturated fatty acids in combination, unsaturated fatty acids alone, and a standard rat chow diet. Twenty rats were in each group. The results show that the rat which consumed caffeine and unsaturated fatty acids had the earliest development of tumor, the most multiple tumor occurrence, and the most rapid rate of growth. Average time to development was 94 days. The fat and water rats had an average time of development of 133 and 140 days respectively. The caffeine alone rats had a mean time to tumor development of 188 days. The combination of an unsaturated fat diet and caffeine significantly shortened the time to tumor development when compared with the other three groups. (no Refs)

10. EXPERIMENTAL INDUCTION OF C CELL TUMOURS IN THYROID BY INCREASED DIETARY CONTENT OF VITAMIN D3.

Thurston V, Williams ED
Dept. Pathology, Welsh Natl. Sch. Medicine, Heath Park, Cardiff CF4 4XN, Wales
Acta Endocrinol (Copenh); 100(1):41-45 1982

Neonatal Wistar rats were given either 0, 5 or 10 uCi 131I within 24 hr of birth. Following weaning, they were fed a diet high, normal or deficient in vitamin D, for up to 2 yr. Animals were sacrificed at approx 3 monthly intervals, and serial sections of thyroid scanned for C cell tumors following calcitonin localization. Plasma calcium levels were also measured, and all results statistically analyzed. As expected, those animals given the high vitamin D had significantly raised calcium levels over those on a normal D diet, whilst those given a low D diet had lower calcium levels than normal. Analysis of the incidence of C cell tumors showed that those given a high D diet had significantly more C cell tumors, whilst those on a low D diet had significantly fewer than normal. Radiation dose also influenced C cell tumor incidence. There was a significant relationship between the vitamin D content of the diet and the incidence of C cell tumors, with those animals on a high D diet having the largest number of tumors. It is suggested that vitamin D or its metabolites may directly promote C cell growth, and that the high incidence of C cell tumors in the normal laboratory rat reflects the artificially high vitamin D content of the laboratory rat diet. The dietary vitamin D content may also be relevant to the variation in geographical incidence of medullary carcinoma in man. We consider it likely that vitamin D metabolites may play a significant role in the control of C cell function, hyperplasia and tumor formation in the rat, and that this may be more important in tumor formation than the role of serum calcium variation. (Author abstract) (20 Refs)

11. CAROTENE AND LUNG CANCER.

Shibusawa K
Natl. Ooji Hosp., Ooji-shi, Japan
Kango; 34(2):75 1982

Epidemiological studies in England, the United States, and Japan have reported that eating foods rich in vitamin A will reduce the risk of having lung cancer. A recent 9-yr long study of the relationship between carotene and retinol consumption and incidence of lung cancer showed that the incidence of lung cancer increased with a drop in the carotene index. The study also indicated that frequency of lung cancer decreased significantly with decreased tobacco consumption and increase carotene consumption. (no Refs) (In JPN)

12. PROMOTION OF LIVER TUMORS IN B6C3F1 MALE MICE BY PARTIAL HEPATECTOMY OR DIETARY CHOLINE DEFICIENCY (MEETING ABSTRACT)

Newberne PM, Clark AJ
Department of Nutrition and Food Science, MIT, Cambridge, MA
Toxicologist; 2(1):63 1982

Groups of male weanling B6C3F1 strain of mice were fed a control semisynthetic diet (C) or the same diet devoid of choline (CD). Some mice from each group were subjected to partial hepatectomy (HepX) after two weeks on diet. Sequential sacrifices of mice from each group revealed the following: after three months, no tumors in any mice although the CD mice had enzyme deficient and fat-free islands of proliferating hepatocytes; after six months choline deficient mice only had liver tumors; after nine months control mice, 0% tumors, C+HepX mice 20% tumors, CD mice 100% tumors and CD+HepX mice 40% tumors. Autoradiographic studies indicated that both HepX and CD increased DNA synthesis and liver cell proliferation at all periods examined. These data suggest that the liver of B6C3F1 mice is sensitive to agents or conditions which are not tumorigens themselves but which may promote tumors. (no Refs)

13. THE PRODUCTION OF URINARY TRACT TUMORS IN RATS BY DIETARY MEANS (MEETING ABSTRACT)

Gershoff SN, Cohen AE, McGandy RB
No affiliation given
77th Annual Meeting of the American Urological Association, Inc., May 16-20, 1982, Kansas City, Missouri. American Urological Association 254 pp., 1982.

In this work retired Charles River CD female breeder rats weighing an average of 370 g were used. The rats were divided into 4 groups and fed purified diets for 9 months with either lactose or sucrose as the carbohydrate. The diets were complete except that half of the animals received diets very low in vitamin A (30 IU of vitamin A palmitate/100 g of diet). Half of the rats fed each carbohydrate had a small stainless steel foreign body inserted into their bladders transuretherally. The foreign bodies rapidly formed a nucleus for the formation of large calculi. On sacrifice all rats fed the low vitamin A diets with either carbohydrate source but none fed adequate levels of the vitamin showed multiple lesions which were characterized as grade I to II transitional cell carcinoma. Many of the lesions as well as other parts of the bladder showed focal areas of squamous metaplasia. In 2 rats there was clear evidence of invasive neoplasms. Many of the rats showed papillomatosis of the renal pelvis. In one case transitional cell carcinoma extensively invading the renal papilla was observed. In conclusion, a method for producing urinary tract tumors in rats has been developed requiring the presence of urinary calculi and chronic vitamin A deficiency. The role of lactose in producing tumors in our previous studies is related to its ability to enhance vesical stone formation. (no Refs)

14. INFLUENCE OF ORAL SUPPLEMENTATION OF ASCORBATE UPON THE INDUCTION OF N-METHYL-N'-NITRO-N-NITROSOGUANIDINE.

Kawasaki H, Morishige F, Tanaka H, Kimoto E
(c/o Kimoto) Dept. Chemistry, Faculty Science, Fukuoka Univ., 11 Nanakuma, Nishi-ku, Fukuoka 814, Japan
Cancer Lett; 16(1):57-63 1982

The influence of oral supplementation of ascorbic acid (AA) upon the induction of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) gastric cancer was investigated in young male Wistar rats. When MNNG-initiated rats (167 ug/ml in the drinking water) were fed a diet containing a daily dose of 0.4 g sodium ascorbate (AA), no significant tumor inhibition was observed, although AA supplementation reduced the grade of the lesion to some extent. When AA supplementation was not begun until 210 days after MNNG, at a time when adenomatous hyperplasia of the stomach epithelium was evident, again, no significant tumor inhibition was observed. AA supplementation in these animals did result in a significant reduction in the grade of the gastric lesions produced, however. Though adenocarcinoma cells seemed to be induced in AA supplemented animals, their downward massive growth, as occurred in animals not supplemented with AA, was inhibited by a heavy proliferation of connective tissue fibers surrounding them. Tumor masses were thus effectively encapsulated by connective tissue in AA supplemented animals. (14 Refs)

15. SELENIUM, ESTRADIOL 17BETA, NITRITES, AND HUMAN BREAST CANCER.

Lombard LF
11145 Northborough Lane, Richmond, VA, 23235
Med Hypotheses; 8(4):371-382 1982

An hypothesis postulating an etiology of breast cancer from interactions among selenium, estradiol 17beta, nitrites, and RBC is presented. In this hypothesis, in the presence of nitrites, triglycerides, and selenium, estradiol 17beta is catalyzed by an unknown enzyme (' X') to form the known carcinogen chrysene, which is complexed with methemoglobin in the RBC. The conversion process may partially take place in the gastrointestinal tract and liver, with more direct conversion in breast cells after transport. Chrysene, in the breast cells, already under attack by nitrites or lysosomal enzymes, causes additional focal hyperplasia, metaplasia, neoplasia, anaplasia, gene alteration, and carcinoma. The process may also require high androgen levels and other trace elements, such as zinc. (51 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

16. EFFECTS OF DIETARY LIPIDS ON LACTOGENIC HORMONE RECEPTOR BINDING IN RAT MAMMARY TUMORS.

Cave WT, Erickson-Lucas MJ
Endocrine Unit, Dept. Internal Medicine, Univ. Rochester Sch. Medicine, St. Mary's Hosp., 89 Genesee St., Rochester, NY, 14611
JNCI; 68(2):319-324 1982

Both epidemiologic studies in humans and experiments in laboratory animals have indicated that high-fat (HF) diets promote mammary tumor growth; however, the biochemical mechanisms responsible for this accelerated tumor growth are poorly understood; thus this study was designed to determine whether diet-induced alterations in the lipid composition of mammary tumor cell membranes were associated with differences in lactogenic hormone binding capacity. Mammary tumors were induced with N-methyl-N-nitrosourea in 50-day-old female inbred Buffalo rats that were maintained on either HF or low-fat (LF) diets composed of either 20% corn oil or 0.5% corn oil, respectively. The microsome-membrane fractions of these tumors were then analyzed for specific lactogenic hormone binding with the use of 125I-labeled human growth hormone. Methylated extracts of these same membrane fractions were also subjected to gas-liquid chromatography. Our results demonstrated that the mammary tumor membranes of the HF group did have a significantly greater lactogenic binding capacity than those of the LF group and that these differences in hormone binding were accompanied by significant alterations in the membrane qualitative fatty acid profiles of each group. Therefore, one way in which dietary lipids may be able to influence mammary tumor growth is by modification of the lactogenic hormone binding capacity of tumor cell membranes. (Author abstract) (27 Refs)

17. CALORIE RESTRICTION: EFFECT ON GROWTH OF HUMAN TUMORS HETEROTRANSPLANTED IN NUDE MICE.

Giovanella BC, Shepard RtC, Stehlin JS, Venditti JM, Abbott BJ
Stehlin Foundation for Cancer Res., 777 St. Joseph Professional Building, Houston, TX, 77002
JNCI; 68(2):249-257 1982

The presence of four human malignant tumors (1 breast, 1 lung, and 2 colon carcinomas) growing sc as heterotransplants in nude mice did not significantly affect the body wt of adult animals until the tumors reached very large dimensions (tumor wt greater than 15% of the body wt). However, a colon carcinoma (HT 29) induced a cessation of the natural rate of body wt increase when it grew in young adults (animals weighing approx 25 g which will gain 6 g or approx 25% body wt in 1 mo). Calorie restriction at all the levels tested (8, 6, 4, and 2 g/day/mouse) with standard pelletized mouse food produced both wt loss in the animals (with and without tumor) and a lowering of the growth rate of all the four tumors tested growing at a sc site and/or under the kidney capsule. Each tumor responded differently to calorie restriction. The four tumors tested grew equally in both male and female nude mice. Young animals weighing 20 g inoculated with a fifth tumor (MeWo melanoma) exhibited tumor growth inhibition proportional to restriction of calorie intake. Their survival, however, did not improve. (Author abstract) (41 Refs)

18. DRUG-NITRITE INTERACTIONS IN HUMAN SALIVA: EFFECTS OF FOOD CONSTITUENTS ON CARCINOGENIC N-NITROSAMINE FORMATION.

Rao GS, Osborn JC, Adatia MR
Div. Biochemistry, Lab. Pharmacology, Res. Inst., American Dental Association Health Foundation, Chicago, IL, 60611
J Dent Res; 61(6):768-771 1982

A simple and rapid high-pressure liquid chromatographic assay for monitoring N-nitrosodimethylamine (NDMA) in human saliva was developed. The method was used to study in vitro the effects of common food constituents on NDMA formation in saliva from the interaction of salivary nitrite with aminopyrine and oxytetracycline. Natural phenolic compounds, caffeic acid, and tannic acid, and synthetic additives, erythorbic acid, sorbic acid, propyl gallate, and butyrated hydroxytoluene - all inhibited NDMA formation (20-80%). With ascorbic acid, up to 90% inhibition of NDMA synthesis in saliva was observed. In contrast, chlorogenic acid (a phenolic component of coffee) acted as a catalyst (up to 48% increase) of the nitrosamine formation under identical experimental conditions. (Author abstract) (21 Refs)

19. PANCREATIC CANCER (MEETING ABSTRACT)

Gold EB, Diener M
Department of Epidemiology, Johns Hopkins University, Baltimore, MD
109th Annual Meeting of the American Public Health Association, November 1-5, 1981, Los Angeles, California. American Public Health Association 390 pp., 1981.

Pancreatic cancer (PC) is a leading cause of death from cancer in the US. Its incidence has been increasing, but little is known of its etiology. A case-control study began in Baltimore in 1978 to investigate factors associated with PC. Over 170 case-neighborhood control pairs have been interviewed. Analysis of the first 107 pairs suggests that dietary carcinogens may be important. More cases than controls frequently ate salami or bologna (matched pairs odds ratio (OR) equals 2.5), bacon or

sausage (OR equals 1.6), eggs (OR equals 2.1), and smoked fish (OR equals 2.1), or beef (OR equals 5). A protective effect was suggested for frequent consumption of high fiber foods such as whole grain breads (OR equals 0.05) and raw fruits or vegetables (OR equals 0.11). A protective effect was suggested for tonsillectomy (OR equals 0.25), also reported for other cancers, but an increased risk was found for living on a farm and having farm animals (OR equals 2.0), and for low income (OR equals 3.8). These preliminary findings suggest that dietary factors may be involved in the etiology of PC. (no Refs)

20. CASE-CONTROL STUDY OF KIDNEY CANCER (MEETING ABSTRACT)

McLaughlin JK, Mandel JS, Blot WJ, Schuman LM
University of Minnesota, Minneapolis, MN
109th Annual Meeting of the American Public Health Association, November 1-5, 1981, Los Angeles, California. American Public Health Association 390 pp., 1981.

A population based interview case-control study of kidney cancer (renal cell 189.0 and renal pelvis 189.1) was conducted in the 7 county Minneapolis St Paul SMSA (standard metropolitan statistical area). Four hundred and ninety-five cases of renal cell and 74 cases of renal pelvis cancer were interviewed (the largest epidemiologic study to date of these sites). Controls (1190) were selected from population based sources and frequency matched on age and sex to cases. Interview response rates of 98% for cases and 97% for controls were achieved. Because of the exploratory nature of the study, many factors were examined including ethnicity and weight, beverage, diet, smoking, drugs, disease and occupational histories. For renal cell cancer the only ethnic group with an elevated risk was German females. Significantly elevated odds ratios were found for males and females for both renal cell and renal pelvis cancer who smoked cigarettes. Males who consumed diet soda had significantly elevated odds ratios. Other elevated odds ratios have been found for certain drugs, industries and occupational exposures. Indices for Vitamins A, C, fat, total alcohol, cruciferous vegetables, phenacetin and acetaminophen have been developed. (no Refs)

21. EFFECT OF DIETARY FIBRE COMPONENTS (CELLULOSE AND LIGNIN) ON EXPERIMENTAL COLON NEOPLASIA (MEETING ABSTRACT)

Sloan DA, Fleiszer DM, Murray D, Brown RA
McGill University Surgical Clinic, Montreal General Hospital, Montreal, Quebec, Canada
Joint Meeting of the Society of Surgical Oncology and Society of Head and Neck Surgeons, April 18-23, 1982, Marco Island, Florida. The Society of Surgical Oncology and the Society of Head and Neck Surgeons 265 pp., 1982.

Considerable interest has been focused on the role of dietary fiber in the etiology of colon cancer, both from epidemiologic and experimental perspectives. Using the well established 1,2-dimethylhydrazine model, we examined the effects of the two major crude fiber components on experimental colon neoplasia. Sixty Sprague-Dawley weanling rats were divided into three dietary groups and pair fed one of three separate but nutritionally identical diets: 'low fiber' (3.8% crude fiber) 'high fiber, cellulose' and 'high fiber, lignin' (both 28.7% crude fiber). The animals were treated with weekly subcutaneous 1,2-dimethylhydrazine injections for twenty weeks at a dose of 20 mgm per kg, and sacrificed at weeks twenty-nine and thirty of the experiment. Both cellulose and lignin fed animals experienced a stool bulking effect with greater 48 hour stool weight than low fiber animals. Animals fed the low fiber diet had significantly longer transit times than the high fiber fed animals. The necropsy results show that only the cellulose (and not the lignin) group had significantly fewer animals with tumor when compared to the low fiber group. The experiment suggests that while cellulose and lignin fibers both have similar stool bulking effects, only cellulose shows a protective effect against the induction of experimental colon cancer. (no Refs)

22. DIETARY FIBER AND CANCER.

Kritchevsky D, Story JA
Wistar Inst. Anatomy and Biology, Philadelphia, PA
Advances in Human Clinical Nutrition. Vitale JJand Breitman SA, ed. Littleton, MA, John Wright - PSG Inc., 217 pp., 1982.

The role of dietary fiber in the development of cancer of the large bowel is discussed. Mechanisms by which a high-fiber diet may protect against colon cancer may involve bile acid metabolism, production of volatile fatty acids through the interaction of intestinal flora with fiber, steroid metabolism, and carcinogens. It is noted that in general, populations ingesting diets high in fiber, do not eat a Western diet. (24 Refs)

23. THE ROLE OF DIETARY FIBER IN LIPID METABOLISM.

Story JA
Dept. Foods and Nutrition, Purdue Univ., West Lafayette, IN
Adv Lipid Res; 18:229-246 1981

The interaction of dietary fiber in lipid metabolism and in lipid-associated disease is discussed. In spite of strong relationships indicated by epidemiological studies, little is known concerning the mechanisms involved and details concerning specific types of dietary fibers involved. The magnitude of the interaction between some types of dietary fiber and bile acids appears sufficient to result in an alteration of bile acid excretion and, as a result, alter sterol balance. At the same time, a resultant increase in concentration of bile acids in the colon may be related to susceptibility to colon carcinogenesis. In one experimental study, the feeding of three types of dietary fiber (alfalfa, cellulose, or bran) led to significantly reduced colon tumors induced by azoxymethane (AOM) when dietary fat was reduced from 35% to 7%. Another study involving use of AOM and intrarectal instillation of methylnitrosourea and various dietary fibers indicated that those fibers which result in dilution of bile acids (eg, bran, cellulose) result in the most consistent reduction in chemically induced colon tumors. (67 Refs)

24. ON THE RELATIONSHIP BETWEEN NUTRITION AND THE DEVELOPMENT OF TUMORS.

Kolb E
Wissenschaftsbereich Tierbiochemie, Karl-Marx-Universitat Leipzig, Semmelweisstrasse 4, DDR-7010 Leipzig, E. Germany
Z Gesamte Inn Med; 37(6):169-176 1982

Chronic intake of carcinogens and cocarcinogens in the diet is a primary factor in the development of tumors in humans. High intake of meats has been linked to cancers of the colon and breast. Reduction in intake of animal fats to less than 50 g/day and increase in intake of raw fibers and carotenes are recommended. Small amounts of smoked meat and fish are not considered to be harmful. Foods containing aflatoxins from fungi should be avoided. (50 Refs) (In GER)

25. METABOLIC EPIDEMIOLOGY OF LARGE BOWEL CANCER.

Hill MJ
Central Public Health Lab., Colindale Ave., London NW9, England
Gastrointestinal Cancer 1. Decosse JJand Sherlock P, ed. The Hague, Martinus Nijhoff Publishers, Cancer Treatment and Research, Vol. 3, 452 pp., 1981.

The epidemiology of large bowel cancer and predisposing and associated diseases are reviewed. Possible causative agents, evidence for the role of bile acids as co-carcinogens, and metabolic epidemiology vis results of studies of the histopathology of large bowel cancer and the adenoma-carcinoma sequence are considered. Variables examined in relation to the development of large bowel cancer include geography, sex, socioeconomic group, race, religion, population density, time and cultural environment. Predisposing diseases include adenomatosis coli, cancer family syndrome, and colorectal adenoma. Associated diseases include associated cancers, diseases of fiber depletion, and others. Possible causative agents are stress and physical factors, radiation, immunological factors, virological factors, environmental agents, endogenous agents (cholesterol, bile acids, steroid estrogens, tryptophan metabolites, tyrosine metabolism, and N-nitroso compounds), nutrition, and genetic factors. (154 Refs)

26. DIETARY HABITS AND COLONIC CARCINOMA.

Bloch R
Klinik fur Verdauungs- und Stoffwechselkrankheiten, Am Weinberg 3-5, D-6430 Bad Hersfeld, W. Germany
Hepatogastroenterology; 28(5):231-232 1981

Dietary factors related to a high incidence of colon cancer (CC) in industrialized countries of Europe and in the United States are discussed; such factors include excesses of fat, protein, meat, and total calories, and deficiencies of fiber, vitamins A, C, and E, and selenium. The ratio of fat to crude fiber has been shown to be more important than the absolute intake of fats and fiber in development of CC. Alcohol, tobacco, appetite-depressing drugs, and laxatives have not been shown to be causative agents in CC. (13 Refs)

27. AETIOLOGICAL ASPECTS OF BLADDER CANCER IN URINARY BILHARZIA.

Mee AD
Urology Unit, Royal Postgraduate Medical Sch., Hammersmith Hosp., London, England
Saudi Med J; 3(2):123-128 1982

The etiology of urinary bladder cancer (BC) is discussed in relation to bilharzia, the incidence of which is much higher in Egypt than in Saudi Arabia. Beta-glucuronidase in the urine has been linked to BC, increased levels of which occur in patients with urinary infections caused by Escherichia coli and Pseudomonas, which are common causes of secondary infections in urinary bilharziasis. However, experimentally, this enzyme has not been proven to be a significant bladder carcinogen. There is experimental evidence that nitrosamines which may form in the urine of some people produce BC. Variations in dietary nitrate could account for the low incidence of BC in the Arabian peninsula and the higher incidence in Egypt. Egyptian farmers have a higher incidence of BC as a complication of bilharzia than professional and business classes. There was also a greater incidence of nitrosamines in the urine of patients with BC and bilharzia than in patients with other cancers of the bladder alone or in patients with bilharziasis alone. The results were not conclusive that nitrosamines were the precipitating cause. The possible role of immunocompetence in urinary bilharziasis is discussed. The present study indicates that carcinoma of the bladder in patients with urinary bilharziasis is due to several factors including carcinogens in the urine of the bladder, and depressed immunocompetence caused by schistosoma antigen. (21 Refs)

PUBLIC POLICY CONCERNING DIETARY ASPECTS OF CARCINOGENESIS

28. INTERACTION OF SAFETY EVALUATION AND GOVERNMENT REGULATIONS (MEETING ABSTRACT)

Kolbye AC
Food and Drug Administration, Washington, DC, 20204
International Conference on Safety Evaluation and Regulation, of Chemicals, February 24-26, 1982, Boston, Massachusetts Bio-Research Institute; 1982

Dr Kolbye will discuss the shortcomings of short-term tests, the problems in defining relevant endpoints for both carcinogenesis and heritable genetic damage and will provide a perspective for the societal needs for improvements in food safety legislation and regulatory practice. Dr Kolbye speaks from the viewpoint of both medicine and law with over 12 years experience in FDA and over 20 years experience with the US government. (no Refs)

29. FOOD ADDITIVES, ALLERGY AND CANCER.

Hill A
Lincoln Coll., Oxford, England
Proc Inst Food Sci Technol [UK]; 15(1):45-50 1982

Regulation of food additives and allergic or carcinogenic responses to food additives were discussed. Basic regulatory guidelines for food additives include (1) usefulness of the substance, satisfying a technological need or a consumer demand, (2) food additives must be safe, as determined by different testing procedures, (3) food additives should not be used in any greater quantities than necessary and the number of additives kept to a minimum, and (4) additives must not mislead the consumer as to the nature or quality of the food. Food additives can and do produce allergic responses (especially urticarial and respiratory reactions) in humans, and reactions can be provoked by testing with several commonly used food colors, preservatives, and antioxidants. Adverse reactions include true allergic or hypersensitivity reactions caused by an immune mechanism, or intolerance where no immunological basis is apparent. Although no measurable contribution to the total cancer incidence by food additives has yet been demonstrated epidemiologically, their possible carcinogenicity has been hotly debated. Research on carcinogenicity of cyclamates, diethylstilbestrol, saccharin, nitrates, and caffeine was discussed. Despite the publicity given to the possible carcinogenicity of food additives, allergic reactions are undoubtedly more common. (20 Refs)

GENERAL REVIEWS AND OTHER STUDIES

30. CANCER: ACHIEVEMENTS, CHALLENGES, AND PROSPECTS FOR THE 1980S. VOLUME 1.

Burchenal JH, Oettgen HF
Memorial Sloan-Kettering Cancer Center, New York, NY
Cancer: Achievements, Challenges, and Prospects for the 1980s. Volume 1. New York, Grune and Stratton, 685 pp., 1981.

The current status of cancer research and prospects for the future are reviewed. Topics include epidemiology (analytic epidemiology, epidemiology and cancer control policy), viruses (polyoma, feline leukemia, sarcoma, Epstein-Barr viruses, retroviruses), radiation hazards (radiation and thyroid, breast, and childhood cancers), industrial and lifestyle carcinogens (metabolic activation of chemical carcinogens, tumor registries), host defense against cancer (immunogenetics of cancer in man and animals, cell-mediated immune reactions to human cancer antigens and their regulation, biochemistry of human tumor antigens), genetics (genetic epidemiology of human cancer, specific fine structural chromosome defects in human neoplasia, clinical features and management of genetic cancers), cell biology and cancer (evolution of the malignant cell phenotype, properties of metastatic tumor cells), prevention of carcinogenesis (inhibitors of chemical carcinogens, advances in the use of retinoids for cancer prevention, blocking of tumor promotion by protease inhibitors), and advances in diagnosis (biochemical and immunologic diagnosis of cancer, advances in nuclear imaging, early diagnosis of liver cancer, advances in diagnosis of gastrointestinal neoplasms). (1641 Refs)

31. TUMORS IN CONTROL HAMSTERS, RATS, AND MICE: LITERATURE TABULATION.

Sher SP
Dept. Safety Assessment, Merck, Sharp, and Dohme Res. Lab., West Point, PA
CRC Crit Rev Toxicol; 10(1):49-79 1982

Rodent bioassays used for evaluation of the carcinogenic potential of drugs or food additives were scanned and data tabulated. Carcinogenic studies greater than 81 wk long in mice and greater than 104 wk long in rats, using concurrent controls, were included. Survival was more variable in hamsters. Recent evidence from laboratories indicates that improved animal husbandry has lengthened survival to greater than 90 wk. The compiled data on tumor incidence were tabulated by species and stock, number of animals, and tumor types. Since chronic carcinogenic studies utilize the hamster, data on this species are included. (99 Refs)

32. EPIDEMIOLOGY OF GASTRIC CANCER AND ITS PRECURSOR LESIONS.

Correa P
Louisiana State Univ. Medical Center, New Orleans, LA, 70112
Gastrointestinal Cancer 1. Decosse JJand Sherlock P, ed. The Hague, Martinus Nijhoff Publishers, Cancer Treatment and Research, Vol. 3, 452 pp., 1981.

Epidemiology of gastric cancer is reviewed with respect to etiology. Topics include geographic distribution, sex ratio, time trends, migrant studies, histopathology, blood groups, socioeconomic class, geochemistry, diet, and pernicious anemia. Historic aspects, histologic characteristics, distribution by sex and age, diet, and nitrate intake are considered in relation to precursor lesions of gastric cancer. (40 Refs)

33. HOST AND ENVIRONMENTAL FACTORS ENHANCING CARCINOGENESIS IN THE RESPIRATORY TRACT.

Nettesheim P, Topping DC, Jamasbi R
Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709
Annu Rev Pharmacol Toxicol; 21:133-163 1981

Selected laboratory studies conducted during the last decade that exemplify what has been learned about the multicomponent etiology of lung cancer are reviewed. Sections concerning experiments relating to modification of host susceptibility include the following: studies on age specific susceptibility of respiratory tract epithelium to carcinogens, effect of immune suppression on the development of respiratory tract tumors, role of dietary vitamin A in the susceptibility to lung cancer induction, and role of respiratory tract infections in the pathogenesis of lung cancer. Sections concerning the interaction of environmental or occupational agents include: two stage carcinogenesis in respiratory tract epithelium, cocarcinogenic effects of chrysotile asbestos, role of so-called inert particles in respiratory tract carcinogenesis, nickel subsulfide as a possible cocarcinogen in occupational lung cancer, studies on benzo(e)pyrene as a cocarcinogen, and role of irritant gases and vapors as cocarcinogens in the respiratory tract. (117 Refs)

34. ANALYSIS OF MUTAGENIC EFFECTS OF FOREIGN SUBSTANCES IN THE FOOD PRODUCTS.

Maganova NB
Inst. Nutrition, Moscow, USSR
Vopr Pitan; (2):3-8 1982

Literature data on the mutagenic hazard of food contamination are reviewed. Food products were found to contain such mutagenic substances as nitrates, nitrites, nitro compounds, salts of heavy metals, pesticides, diethylstilbestrol, and aflatoxins. The need for a comprehensive mutagenicity testing of food products is emphasized. (82 Refs) (In RUS)

35. CAFFEINE: AN UPDATE.

Oser BL, Ford RA
Bernard L. Oser and Associates, 108-18 Queens Blvd., Forest Hills, NY, 11375
Drug Chem Toxicol; 4(4):311-329 1981

Intake patterns, pharmacokinetics and metabolism, reproduction and teratology, behavioral effects, and epidemiology of caffeine as a component of beverages are reviewed. Reproductive and teratologic studies are questioned with respect to the applicability of animal studies to humans. No evidence for carcinogenicity of caffeine in long-term studies has been published. No available epidemiological study reveals convincing evidence of carcinogenicity, teratogenicity, or adverse behavioral effects from caffeine ingestion in soft drinks, coffee, or tea. (54 Refs)

36. INHIBITION OF N-NITROSAMINES IN BACON.

Gray JI, Reddy SK, Price JF, Mandagere A, Wilkens WF
Dept. Food Science and Human Nutrition, Michigan State Univ., East Lansing, MI, 48824
Food Technol; 36(6):39-45 1982

Mechanisms of N-nitrosamine formation during cooking of bacon, approaches to reduction of N-nitrosamine levels, and inhibition of N-nitrosation reactions are reviewed, and the applicability and effectiveness of alpha-tocopherol-coated salts in inhibiting N-nitrosamine formation in both dry-cured and brine-

cured bacons are discussed. Alpha-tocopherol-coated salt systems are very effective inhibitors of N-nitrosamine formation in fried bacon, and do no affect the safety of the product or the antibotulinal activity of nitrite. (38 Refs)

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. TOXICITY OF POLYBROMINATED BIPHENYLS (PBBS) IN DOMESTIC AND LABORATORY ANIMALS.

Damstra T, Jurgelski W, Posner HS, Vouk VB, Bernheim NJ, Guthrie J, Luster M, Falk HL
Natl. Inst. Environmental Health Sciences, Post Office Box 12233, Research Triangle Park, NC, 22709
Environ Health Perspect; 44():175-188 1982

2. CHLOROFORM: A REVIEW OF ITS METABOLISM, TERATOGENIC, MUTAGENIC, AND CARCINOGENIC POTENTIAL.

Davidson IW, Sumner DD, Parker JC
(c/o Parker) ECAO, Environmental Protection Agency, MD-52, Research Triangle Park, MD, 27711
Drug Chem Toxicol; 5(1):1-87 1982

3. FORMALDEHYDE IN PRESENT-DAY ENVIRONMENTAL TOXICOLOGY.

Beritic T, Kovac S, Dimov D
Odjel za profesionalne bolesti, Institut za medicinska istrazivanja i medicinu rada, Zagreb, Yugoslavia
Arh Hig Rada Toksikol; 32(4):363-395 1981
In SCR

4. HORMONES AS ETIOLOGICAL AGENTS IN NEOPLASIA.

Furth J
Inst. Cancer Res., Columbia Univ. Coll. Physicians and Surgeons, New York, NY
Cancer - A Comprehensive Treatise Vol. 1. Etiology - Chemical and Physical Carcinogenesis. Second Edition. Becker FF, ed. New York, Plenum Press, 714 pp., 1982.

5. CURRENT VIEWS ON HORMONES AND BREAST CANCER.

Molinatti GM, Limone P
Cattedra di Endocrinologia, Universita di Torino, C.so Polonia, 14, 10126 Turin, Italy
Minerva Med; 73(17):1047-1053 1982
In ITA

6. THE CHROMOSOMAL EFFECTS OF THE ORAL CONTRACEPTIVE.

Bixbus NF
Tissue Culture and Cytogenetics Unit, Res. Dept., Marie Curie Memorial Foundation, The Chart. Oxted, Surrey, England
J Surg Oncol; 20(2):115-118 1982

7. TOXICITY AND CARCINOGENICITY OF NICKEL AND ITS COMPOUNDS. A REVIEW OF THE CURRENT STATUS.

Raithel HJ, Schaller KH
Institut fur Arbeits- und Sozialmedizin, Poliklinik fur Berufskrankheiten, Universitat Erlangen-Nurnberg, Schillerstrasse 25-29, D-8520 Erlangen, W. Germany
Zentralbl Bakteriol Mikrobiol Hyg [B]; 173(1/2):63-91 1981
In GER

8. HISTORICAL OVERVIEW OF NITROSOUREA DEVELOPMENT.

Goldin A
Div. Cancer Treatment, NCI NIH, Building 37, Room 5E20, Bethesda, MD, 20205
INSERM Symp; 19:3-11 1981

9. PATHOLOGY.

Goyer RA
Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC
JAMA; 247(21):2973-2975 1982

RADIATION CARCINOGENESIS

10. IDENTIFYING THE HEALTH RISKS FROM VERY LOW-DOSE SPARSELY IONIZING RADIATION.

Dreyer NA, Friedlander E
Epidemiology Resources, Inc., P.O. Box 57, Chestnut Hill, MA, 02167
Am J Public Health; 72(6):585-588 1982

11. RISK OF RADIOLOGICAL EXAMINATIONS.

Jankowski J
Instytut Medycyny Pracy w Lodzi, ul. Olowiana 6, 91-614 Lodz, Poland
Pol Przegl Radiol; 45(6):383-386 1981
In POL

12. RADIOBIOLOGY OF RISK ESTIMATES IN MEDICAL IRRADIATION (MEETING ABSTRACT)

Rossi HH
Columbia University, New York, NY
IN: 148th National Meeting of the American Association for the Advancement of Science, January 3-8, 1982, Washington, D.C American Association for the Advancement of Science 182 pp., 1982.

13. RADIATION-INDUCED VASCULAR INJURY AND ITS RELATION TO LATE EFFECTS IN NORMAL TISSUES.

Law MP
MRC Cyclotron Unit, Hammersmith Hosp., London W12 OHS, England
Adv Radiat Biol; 9:37-73 1981

14. CURRENT CONCEPTS IN ULTRAVIOLET CARCINOGENESIS.

Grantstein RD, Sober AJ
(c/o Sober) Dept. Dermatology, Massachusetts General Hosp., Boston, MA, 02114
Proc Soc Exp Biol Med; 170(2):115-125 1982

15. METABOLIC CHANGES AFTER EXPOSURE TO RADIONUCLIDES.

Kalistratova VS
No affiliation given
Med Radiol (Mosk); 27(4):62-71 1982
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DIETARY FACTORS IN CARCINOGENESIS

16. CAFFEINE: AN UPDATE.

Ower BL, Ford RA
Bernard L. Ower and Associates, 108-18 Queens Blvd., Forest Hills, NY, 11375
Drug Chem Toxicol; 4(4):311-329 1981

17. ANALYSIS OF MUTAGENIC EFFECTS OF FOREIGN SUBSTANCES IN THE FOOD PRODUCTS.

Maganova NB
Inst. Nutrition, Moscow, USSR
Vopr Pitan; (2):3-8 1982
In RUS

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

18. RADIATION EPIDEMIOLOGY (MEETING ABSTRACT)

Marks S
No affiliation given
148th National Meeting of the American Association for the Advancement of Science, January 3-8, 1982, Washington, D.C. American Association for the Advancement of Science 182 pp., 1982.

19. EPIDEMIOLOGICAL STUDIES ON RADIATION CARCINOGENESIS IN HUMAN POPULATIONS FOLLOWING ACUTE EXPOSURE: NUCLEAR EXPLOSIONS AND MEDICAL RADIATION.

Fabrikant JI
Donner Lab., Univ. California, Berkeley, CA, 94720
Yale J Biol Med, 54(6):457-469 1981

20. EPIDEMIOLOGIC APPROACH TO CANCER.

Guinee VF
Dept. Epidemiology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX
Cancer - A Comprehensive Treatise Vol. 1. Etiology - Chemical and Physical Carcinogenesis. Second Edition. Becker FF, ed. New York, Plenum Press, 714 pp., 1982.

21. EPIDEMIOLOGY AND EARLY DETECTION OF CANCER OF THE ESOPHAGUS.

Sorouri P
900 Hillside Road, Wilmington, DE, 19807
Gastrointestinal Cancer 1. Decosse JJand Sherlock P, ed. The Hague, Martinus Nijhoff Publishers, Cancer Treatment and Research, Vol. 3, 452 pp., 1981.

22. METABOLIC EPIDEMIOLOGY OF LARGE BOWEL CANCER.

Hill MJ
Central Public Health Lab., Colindale Ave., London NW9, England
Gastrointestinal Cancer 1. Decosse JJand Sherlock P, ed. The Hague, Martinus Nijhoff Publishers, Cancer Treatment and Research, Vol. 3, 452 pp., 1981.

23. EPIDEMIOLOGY OF GASTRIC CANCER AND ITS PRECURSOR LESIONS.

Correa P
Louisiana State Univ. Medical Center, New Orleans, LA, 70112
Gastrointestinal Cancer 1. Decosse JJand Sherlock P, ed. The Hague, Martinus Nijhoff Publishers, Cancer Treatment and Research, Vol. 3, 452 pp., 1981.

24. TRENDS IN THE INCIDENCE OF GASTRIC CARCINOMA (MEETING ABSTRACT)

Nobrega FT
Preventive and Internal Medicine, Mayo Clinic, Rochester, MN
Eleventh Annual William Beaumont Gastrointestinal Symposium, March 24-26, 1982, El Paso, Texas. William Beaumont Army Medical Center 56 pp., 1982.

25. CANCER IN INFLAMMATORY BOWEL DISEASE.

Bayless TM, Yardley JH
Johns Hopkins Hosp., Baltimore, MD, 21205
Gastrointestinal Cancer 1. Decosse JJand Sherlock P, ed. The Hague, Martinus Nijhoff Publishers, Cancer Treatment and Research, Vol. 3, 452 pp., 1981.

26. GENETIC ASPECTS OF GUT CANCER.

McConnell RB
Broadgreen Hosp., Liverpool, England
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27. HOST AND ENVIRONMENTAL FACTORS ENHANCING CARCINOGENESIS IN THE RESPIRATORY TRACT.

Nettesheim P, Topping DC, Janssbi R
Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709
Annu Rev Pharmacol Toxicol; 21:133-163 1981

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28. A SEARCH FOR SAFE DRINKING WATER.

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A Search for Safe Drinking Water. Available from National Technical Information Service, Springfield, VA, as HRP-0903845/6, 68 pp., 1981.

29. OCCUPATIONAL EXPOSURES TO URANIUM: PROCESSES, HAZARDS, AND REGULATIONS.

Stoetzel GA, Fisher DR, McCormack WD, Hoenes GR, Marks S
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Occupational Exposures to Uranium: Processes, Hazards, and Regulations. Available from National Technical Information Service, Springfield, VA, as PNL-3341-USUR-01:, 91 pp., 1981.

CELLULAR AND MOLECULAR MECHANISMS IN CARCINOGENESIS

30. THE ROLE OF RADIATION AND CHEMICALS IN THE INDUCTION OF MUTATIONS AND EPIGENETIC CHANGES DURING CARCINOGENESIS.

Trosko JE, Chang CC
Dept. Human Development, Coll. Human Medicine, Michigan State Univ., East Lansing, MI, 48824
Adv Radiat Biol; 9:2-36 1981

31. SOME EFFECTS OF CARCINOGENS ON CELL ORGANELLES.

Svoboda DJ, Reddy J
Dept. Pathology and Oncology, Univ. Kansas Coll. Health Sciences, Kansas City, KS
Cancer - A Comprehensive Treatise Vol. 1. Etiology - Chemical and Physical Carcinogenesis. Second Edition. Becker FF, ed. New York, Plenum Press, 714 pp., 1982.

32. CHEMICAL CARCINOGENESIS: INTERACTIONS OF CARCINOGENS WITH NUCLEIC ACIDS.

Rajalakshmi S, Prema M, Sarma D
Dept. Pathology, Univ. Toronto, Toronto, Ontario, Canada
Cancer - A Comprehensive Treatise Vol. 1. Etiology - Chemical and Physical Carcinogenesis. Second Edition. Becker FF, ed. New York, Plenum Press, 714 pp., 1982.

33. A MOLECULAR RATIONALE FOR NITROSOUREA INDUCED CYTOTOXICITY.

Tew KD
Lombardi Cancer Center, Div. Medical Oncology, Georgetown Univ., 3800 Reservoir Road, N.W., Washington, DC, 20007
INSERM Symp; 19:61-77 1981

34. MODIFICATION OF DNA AND RNA BASES BY THE NITROSOUREAS.

Ludlum DB, Tong WP
Div. Oncology, Dept. Pharmacology and Experimental Therapy, Albany Medical Coll., Albany, NY, 12208
INSERM Symp; 19:21-31 1981

35. CALMODULIN AND CA2+ IN NORMAL AND TRANSFORMED CELLS.

Veigl ML, Sedwick WD, Vanaman TC
Dept. Microbiology and Immunology, Duke Univ. Medical Center, Durham, NC, 27710
Fed Proc; 41(7):2283-2288 1982

36. METABOLISM AND TOXICITY OF AFLATOXINS.

Hsieh DP, Wong JJ
Dept. Environmental Toxicology, Univ. California, Davis, Davis, CA, 95616
Adv Exp Med Biol; 136(partB):847-863 1982

37. REACTION OF MICROSOMAL AND CYTOSOLIC ENZYMES WITH N-ARYLACETOHYDROXAMIC ACIDS IN THE PRESENCE OF OXYGEN.

Lenk W, Scharmer U
Pharmakologisches Institut, Universitat Munchen, Nussbaumstrasse 26, D-8000 Munchen 2, W. Germany
Adv Exp Med Biol; 136(partB):951-970 1982

38. METABOLIC ACTIVATION AND DEACTIVATION MECHANISMS OF DI-, TRI-, AND TETRACHLOROETHYLENES.

Henschler D, Hoos R

Inst. Toxicology, Univ. Wurzburg, Versbacher Strasse 9, Wurzburg D-8700, W. Germany

Adv Exp Med Biol; 136(partA):639-666 1982

39. METABOLIC ACTIVATION AND HEPATOTOXICITY OF TRICHLOROETHYLENE.

Reynolds ES, Mcalen MT

Chemical Pathology Lab., Dept. Pathology, Univ. Texas Medical Branch, Galveston, TX

Adv Exp Med Biol; 136(partA):693-701 1982

40. THE ACTIVATING ROLE OF GLUTATHIONE IN THE MUTAGENICITY OF 1,2-DIBROMOETHANE.

van Bladeren PJ, Breimer DD, Rotteveel-Smijs GM, Hoogsteep JJ, Mohn GR, De Groot A, Van Zeeland AA, Van der Gen A

Dept. Pharmacology, Univ. Leiden, P.O. Box 9502, 2300 RA Leiden, The Netherlands

Adv Exp Med Biol; 136(partA):809-820 1982

41. METABOLISM OF POLYCYCLIC AROMATIC HYDROCARBONS IN PLACENTA.

Jachau MR, Chao ST, Namkung MJ

Dept. Pharmacology, Sch. Medicine, SJ-30, Univ. Washington, Seattle, WA, 98195

Adv Exp Med Biol; 136(partA):555-573 1982

MODIFICATION OF CARCINOGENESIS

42. THE INTESTINAL ENZYMATIC BIOTRANSFORMATION SYSTEM: POTENTIAL ROLE IN PROTECTION FROM COLON CANCER.

Hoensch HP, Hartmann F

Medizinische Universitatsklinik, D-7400 Tubingen, W. Germany

Hepatogastroenterology; 28(4):221-228 1981

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

43. THE ONCOGENIC CIRCLE CLOSES.

Rigby PW

Imperial Coll. Science and Technology, London SW7 2BZ, England

Nature; 297(5866):451-453 1982

44. ANALYSIS OF CASES OF HEPATIC CELL CARCINOMA THAT TRANSFORMED FROM VIRAL HEPATITIS.

Ichida F, Honma A, Soga K

Third Dept. Internal Medicine, Niigata Univ. Sch. Medicine, Niigata, Japan

Nippon Rinsho; 40(2):468-477 1982

In JPN

45. ROLE OF VIRUSES IN THE INDUCTION OF PRIMARY INTRACRANIAL TUMORS.

Welsh JW, Zimmer SG, Perdue ML

Div. Neurosurgery, Univ. Kentucky Medical Center, 800 Rose St., Lexington, KY, 40536

Neurosurgery; 10(5):643-662 1982

METHODOLOGY IN CARCINOGENESIS RESEARCH

46. TRANSLATION OF PHARMACOKINETIC/BIOCHEMICAL DATA INTO RISK ASSESSMENT.

Dietz FK, Reitz RH, Watanabe PG, Gehring PJ

Toxicology Res. Lab., Health and Environmental Sciences, Dow Chemical, 1803 Building, Midland, MI, 48640

Adv Exp Med Biol; 136(partB):1399-1424 1982

47. IN VITRO ASSAYS FOR THE DIFFERENTIATION OF TUMOR PROMOTORS FROM CARCINOGENS AFTER ROUTINE CARCINOGENESIS BIOASSAYS (MEETING ABSTRACT)

Ward JM, Rice JM

Frederick Cancer Research Center, Frederick, MD

Second Annual Meeting of the Society of Environmental Toxicology and Chemistry, November 22-25, 1981, Arlington, Virginia. Society of Environmental Toxicology and Chemistry 150 pp., 1982.

48. ENVIRONMENTAL CARCINOGENS: SELECTED METHODS OF ANALYSIS. VOLUME 4. SOME AROMATIC AMINES AND AZO DYES IN THE GENERAL AND INDUSTRIAL ENVIRONMENT.

Egan H, Fishbein L, O'Neill IK, Castegnaro M, Bartsch H

Lab. Government Chemist, London, England

IARC Sci Publ (40): 347 pp., 1981.

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DIETARY ASPECTS OF CARCINOGENESIS

SERIES CK03 No. 82/11 NOVEMBER, 1982

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- GENERAL REVIEWS AND OTHER STUDIES..... 22-27

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

1. THE HEALTH EFFECTS OF NITRATE, NITRITE AND N-NITROSO COMPOUNDS: PART 1 OF A 2-PART STUDY.

National Research Council
Washington, DC
The Health Effects of Nitrate, Nitrite, and N-Nitroso Compounds: Part 1 of a 2-Part Study. Available from National Technical Information Service, Springfield, VA, as PB82-182890, 534 pp., 1981.

The report contains an evaluation of the health effects of nitrate, nitrite and N-nitroso compounds. The major conclusions of this report are that there is not sufficient evidence to suggest that nitrate and nitrite are carcinogenic in animals, and epidemiological studies are also inconclusive. However, nitrate and nitrite can be converted in foods and in the body to nitroso compounds which are carcinogenic in animals and possibly in humans. The added risk for nitrate and nitrite added to foods is small except in special populations consuming large amounts of cured meats. Vitamins C and E were found to be effective inhibitors of nitrosation. The report recommends reducing human exposure to nitrate, nitrite, and N-nitroso compounds,

HE 20.3173/2:

pursuing research on substitutes for nitrite and on antioxidants (eg, vitamins C and E) that can block nitrosation. (Author abstract)

2. NITRATE IN DRINKING WATER AND CANCER IN NORTHERN JUTLAND, DENMARK, WITH SPECIAL REFERENCE TO STOMACH CANCER.

Jensen OM
Danish Cancer Registry, Strandboulevarden 49, DK-2100 Copenhagen 0, Denmark
Ecotoxicol Environ Saf; 6(3):258-267 1982

Cancer risk in an area with long-term evidence of nitrate in drinking water was studied, and cancer morbidity was compared with that in neighboring towns with zero or low levels of nitrates in water, and in Danish provincial towns. A slight but significant increased risk of developing cancer of the stomach was found in Aalborg compared to neighboring towns and provincial towns; the increase was consistent over the period 1943-1976. A decrease in stomach cancer was noted in all places studied over the period examined. (33 Refs)

3. POLYCYCLIC AROMATIC HYDROCARBONS IN SMOKED FISH.

Larsson BK
Food Lab., Natl. Food Admin., Box 622, S-751 26 Uppsala, Sweden
Z Lebensm Unters Forsch; 174(2):101-107 1982

Concentrations of polycyclic aromatic hydrocarbons were measured in 70 samples of smoked fish and fish products. The samples had been produced by a variety of smoking methods. Concentrations of benzo[a]pyrene (BaP) exceeded 1 ug/kg in 19/46 samples of commercially smoked fish and 4/16 samples of canned smoked fish. BaP levels were below 1 ug/kg when fish were smoked using external smoke generators. (15 Refs)

4. AFLATOXIN IN FOODS OF VEGETABLE AND ANIMAL ORIGIN

Horvath E, Biro Za, Adrassy K, Horvath I
Megyei Kozegeszsegugyi-Jarvanyugyi Allomas, Debrecen, Hungary
Orv Hetil; 123(20):1235-1239 1982

Aflatoxin B1 levels of 0.005 mg/kg and higher were found in 35/96 samples of imported peanuts in Hajdu-Bihar county, Hungary, during the period 1976-1979. Aflatoxin was found in 4/80 whole milk samples, 1/40 milk powder samples and 0/40 egg samples investigated during the same period. (34 Refs) (In HUN)

5. INTRAUTERINE EXPOSURE TO SACCHARIN AND RISK OF CANCER OF THE URINARY BLADDER IN HUMANS.

Kamby C
Cancerregisteret, Strandboulevarden 49, DK-2100 Copenhagen 0, Denmark
Ugeskr Laeger; 144(20):1480-1482 1982

The incidence of cancer of the urinary bladder was studied on the birth cohort 1941-1945 compared with the birth cohorts 1931-1935 and 1936-1940 (all in the age bracket 20-34 yr) in view of the evidence of increased risk of bladder cancer due to intrauterine exposure to saccharin in experimental animals and to the increased consumption of saccharin during the period 1941-1945. The birth cohort 1941-1945 included 996,866 men and 947,590 women in the age group 20-24 yr, 993,963 men and 942,257 women in the age group 25-29 yr and 467,461 men and 444,503 women in the age group 30-34 yr. No significant differences were found between the observed and expected numbers of cancer of the urinary bladder in the birth cohort 1941-1945 compared with the two other birth cohorts. The relative risk of bladder cancer of the men in the birth cohort 1941-1945 was identical (1.0) with that of the men in the birth cohorts 1931-1935, 1936-1940 and 1931-1940. The relative risk of bladder cancer in the women of the birth cohort 1941-1945 was reduced (0.3) compared with that of the women in the two previous cohorts. The findings indicate that massive intrauterine exposure to saccharin did not involve any risk of cancer of the urinary bladder during the first 20-34 yr of life. (15 Refs) (In DAN)

EXPERIMENTAL STUDIES OF POTENTIAL CARCINOGENS IN THE DIET

6. MUTAGENICITY OF HYDROLYSATES OF CITRUS FRUIT JUICES.

Mazaki M, Ishii T, Uyeta M
Public Health Inst. Kochi Prefecture, 2-4-1, Marunouchi, Kochi 780, Japan
Mutat Res; 101(4):283-291 1982

Hydrolysates of citrus fruit juices obtained with acid, enzymes, and human intestinal bacteria were tested for mutagenicity in the Ames test using strains TA98 and TA100. The hydrolysates of all citrus fruit juic canned orange juice, fresh fruit juice of mandarin orange, grapefruit, navel orange, 'Ponkan', and pomelo used in the study were mutagenic. Mutagenicities were found in both the chloroform extract and the ether extract. The chloroform extracts were mutagenic to TA98 and TA100 with S9 mix, and the ether extracts were mutagenic to TA98 and TA100 with or without S9 mix. (19 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

7. DIET IN THE EPIDEMIOLOGY OF ORAL CANCER.

Marshall J, Graham S, Mettlin C, Shedd D, Swanson M
Dept. Social and Preventive Medicine, State Univ. New York at Buffalo, Buffalo, NY
Nutr Cancer; 3(3):145-149 1982

In this comparison of 425 oral cancer patients and 588 controls, it was found that risk increased in dose-response fashion as vitamin A or vitamin C intake decreased. This elevation of relative risk persisted with control for smoking and alcohol ingestion. (Author abstract) (15 Refs)

8. EFFECT OF VITAMIN A AND ANALOGS ON THE INDUCTION OF SQUAMOUS CELL CARCINOMA IN HAIRLESS MICE CAUSED BY SOLAR SIMULATED UV-LIGHT (MEETING ABSTRACT).

Israili ZH, Razdan R, Willis I
Emory University, Atlanta, GA, 30322
Proc Am Assoc Cancer Res; 23:806 1982

The UV component of the sunlight is the primary stimulus for the majority of skin cancers, the most common of human

malignancies. Vitamin A and analogs have been shown to prevent certain chemical carcinogen-induced cancers. We tested the effectiveness of vitamin A and analogs as chemopreventive and/or therapeutic agents in a hairless mouse model in which squamous cell carcinoma in the skin is induced by irradiation with solar stimulating UVA (320-400 nm) plus UVB (290-320 nm) in an escalating dose (9.0-15.3 J/cm2, daily, 5 days/wk for 8 wks). Groups of female mice (12-16 wks old) were irradiated and treated as follows: a) N equals 50, controls, b) N equals 46, all-trans-retinoic acid (t-RA, 20 mg/kg body weight), 8 wks during plus 13 wks post-UV, ie, 21 wks, c) N equals 19, t-RA, 13 wks post-UV, d) N equals 10, 13-cis-retinoic acid (c-RA, 80 mg/kg), for 21 wks, e) N equals 10, c-RA, 13 wks post-UV, f) N equals 10, retinol (33 mg/kg), for 21 wks, and g) N equals 10, beta-carotene (33 mg/kg), for 21 wks. Vitamin A and analogs, as suspensions (in 0.3 ml of 1:1 v/v of Carnation milk:water), were given orally 3 times per wk. Mice were maintained on Purina mouse chow and water ad lib, and were observed for 26 wks. No tumors appeared in unirradiated mice given vitamin A analogs. No significant difference was found in the incidence of cancer (papilloma and tumor) at 26 wk in any of the groups of irradiated animals: a) 20/50, b) 16/43, c) 7/16, d) 3/6, e) 4/9, f) 3/8, and g) 5/8. Retinoyl palmitate (300 mg/kg in 0.2 ml corn oil) given orally, 3 times/wk for 6 wks, to 4 mice with large tumors caused some decrease in tumor size. No histological differences were observed in any of the tumors. (no Refs)

9. PREVENTION AND REVERSAL BY A RETINOID OF 3,4-BENZPYRENE- AND CIGARETTE SMOKE CONDENSATE-INDUCED HYPERPLASIA AND METAPLASIA OF RODENT RESPIRATORY EPITHELIA IN ORGAN CULTURE.

Lasnitzki I, Bollag W
Strangeways Res. Lab., Worts Causeway, Cambridge, CB1 4RN, England
Cancer Treat Rep; 66(6):1375-1380 1982

The influence of an aromatic analog of vitamin A, etretinate, on the effects of 3,4-benzpyrene and cigarette smoke condensate has been investigated in fetal mouse lung and neonatal rat tracheas grown in organ culture. In both tissues, 3,4-benzpyrene as well as cigarette smoke condensate induces a striking increase of epithelial mitosis within 12-14 days of treatment. The increase is associated with a loss of secretory activity and of ciliary function. These changes persist in the absence of benzpyrene or smoke condensate in explants transferred to control medium. Treatment with etretinate alone does not affect the normal epithelial growth rate or normal differentiation. If combined with either benzpyrene or smoke condensate, the aromatic compound inhibits the increase in cell division and prevents the loss of secretory activity or ciliary function. In explants pretreated with 3,4-benzpyrene or cigarette smoke condensate, etretinate reduces the carcinogen- or smoke condensate-induced increase in mitotic activity to normal levels and restores secretory differentiation and ciliary function. The mechanism of action involved in the anticarcinogenic activity of the retinoid is discussed. (Author abstract) (32 Refs)

10. SELENIUM ENHANCED CONJUGATION OF BENZO(A)PYRENE BY HEPATOCYTES (MEETING ABSTRACT).

Marshall MV, Gonzalez MA, Griffin AC
Southwest Foundation for Research and Education, San Antonio, TX, 78284
Proc Am Assoc Cancer Res; 23:278 1982

Previous studies have attributed anticarcinogenic properties to enhanced dietary intake of selenium. No mechanism of action has yet been determined, although several reports have demonstrated enhanced detoxification of chemical carcinogens requiring metabolic activation. The purpose of this study was to investigate the detoxification of benzo(a)pyrene (BaP) by hepatocytes isolated from rats fed a selenium supplementation of 4 ppm in their drinking water. Rats were fed a control diet or one containing 4 ppm Se (as Na2SeO(3)) for 15 days. After 11 days on the experimental diet, animals in both groups (control, Se) were injected with Aroclor 1254 to induce mixed function oxidase activity. Hepatocytes were isolated 4 days later and cultured with 50 uM 3H BaP for up to 2 h. In hepatocytes isolated from the Se group, an increase in sulfate, glucuronide, and glutathione conjugation was observed. An increase in dihydrodiol and phenol formation and a decrease in quinone formation was also observed for the free metabolites. In summary, dietary Se administration enhanced the conjugation of BaP. Increases in metabolism were also observed in the free metabolite fraction. The increase in dihydrodiol production also implicates an enhancement of epoxide hydrolase activity. The overall effect was to increase conversion of BaP to water soluble metabolites and conjugates. (no Refs)

11. A CASE CONTROL STUDY OF SKIN NEOPLASMS AND THE ANTICARCINOGENIC EFFECT OF SELENIUM.

Clark LC
The University of North Carolina at Chapel Hill, Chapel Hill, NC
Diss Abstr Int [B]; 42(12,pt1):4753-B 1982

This case control study tested the hypothesis that plasma selenium levels would be lower in patients with skin cancer than in age-sex strata matched controls. The study included 240 cases of skin cancer with a diagnosis of basal cell epithelioma (142) and/or squamous cell epithelioma (98), and two control groups, current clinic controls (60) and random clinic controls (43). Patients with low selenium levels had a consistent increase in risk of skin cancer for each histologic tumor type compared to either control group. The logistic estimate of the odds ratio for the highest versus the lowest decile of selenium for all cases and the clinic controls is 4.39; for all cases and the random controls the logistic estimate of the odds ratio is 5.81. This association was consistent in every analysis, and was stronger in younger patients and in patients with low sun damage. The clinical assessment of sun damage was the most predictive variable for skin cancer in all of the logistic regression models. Patients with a history of arsenic exposure were also at an increased risk of having skin cancer. The association of skin cancer with plasma vitamin A and beta carotene levels was confounded by a nutritional counseling program at the clinic, by the use of vitamin supplements, and by a seasonal trend in vitamin A levels. Vitamin A was a significant protective factor for skin cancer for the logistic models with random controls. The lack of consistency for this protective effect may be due to confounding. Beta carotene was associated with an increased risk of skin cancer in all of the logistic analyses; however this association may be spurious due to confounding as a result of a nutritional counseling program. (no Refs)

12. DAUNORUBICIN (D) INDUCED MAMMARY TUMORS IN FEMALE SPRAGUE-DAWLEY RATS AND THE ROLE OF ALPHA-TOCOPHEROL (ALPHAT) AS A MODIFIER TO THE CARCINOGENESIS (MEETING ABSTRACT).

Wang YM, Howell SK, Tsai CC, Van Eys J
M. D. Anderson Hospital and Tumor Institute, Houston, TX, 77030
Proc Am Assoc Cancer Res; 23:352 1982

Second malignancies are significant side effects of cancer chemotherapy. Anthracycline antibiotics-induced tumors have been recognized in rats and monkey. We have, recently, investigated the activity of alphaT as a potential modifier to the carcinogenecity-induced by D in female rats. Two groups of rats at age 40 day (less than 125 g) and 48-56 days (150-200 g) respectively were used. In older animals, D (10 mg/kg) injected intravenously induced tumors in 50% (11/22) of the animals. Preinjection with alpha-tocopheryl acetate (alphaTA, 300 mg/kg/day) daily for consecutive 4 days prior to the injection of D reduced significantly the tumor incidence (2/16). In younger animals, tumor incidence was less (5/21) and the pretreatment with the vitamin did not modify the tumor development (9/27). In older animals, organ distribution of 3H-D and 14C-alphaTA changes by each other, however, alphaTA does not alter the concentration of D in mammary tissue and fat. However, in single mammary epithelial cells suspensions isolated in rats after 4 daily treatment of alpha-TA or placebo, the intracellular D distribution was significantly modified in the presence of alphaT and alphaTA. The concentration of 3H-D in nuclear fractions was 30-40% in those cells isolated from alphaT treated animals as compared to the cells from placebo treated animal. It appears the alteration of subcellular D distribution by the vitamin may play a role in the modification of D-induced mammary tumor in rats. (no Refs)

13. EFFECT OF A MARGINALLY LIPOTROPE-DEFICIENT DIET ON METHYLAZOXYMETHANOL ACETATE CARCINOGENESIS.

Kato K, Mori H, Takahashi M, Hirono I
Dept. Pathology, Gifu Univ. Sch. Medicine, Gifu, Japan
Nutr Cancer; 3(4):234-239 1982

The effect of a lipotrope-deficient diet on methylazoxymethanol acetate (MAM acetate) intestinal carcinogenesis was studied in 2 groups of ACI rats. For 10 wk, weanling rats were fed a semisynthetic diet that was either high in fat and marginally deficient in the lipotropes (Group 1), or adequate for rats in all known respects (Group 2). Four wk after the start of the experimental diets, the animals were given a weekly SC injection of MAM acetate, 25 mg/kg body weight, for a total of 5 times. Following the termination of the experimental diets, the animals were fed a basal diet until the end of the experiment (480 days). The tumor incidence in the jejunum-ileum and the number of tumors in the colon were significantly higher in Group 1 than in Group 2. These results suggest that a marginally lipotrope-deficient diet enhances MAM acetate carcinogenesis, probably in its early stage. (Author abstract) (14 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

14. DIETARY PRACTICES OF EARLY LIFE AND SPONTANEOUS TUMORS OF THE RAT.

Ross MH, Lasthader ED, Bras G
Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA, 19111
Nutr Cancer; 3(3):150-167 1982

A complete life history was obtained of the dietary practices, growth responses, and diseases of outbred male Charles River COBS rats permitted to select their own diets. Simple correlation analyses proved inadequate for detecting an association between any single factor at any age and tumor development. A multifactorial model showed that animals that developed tumors could be distinguished from those that did not, solely on the basis of age- or weight-specific information prior to maturity. The combination of variables that maximize the probability of a neoplasm is: (1) a high absolute protein intake shortly after weaning, (2) a high degree of efficiency in converting consumed food into body mass at the time the individual enters puberty, (3) a high level of protein intake relative to body wt during the early adult period and concomitantly, (4) a high level of food intake, and (5) a rapid growth rate during early postnatal life so that comparatively less time is required to attain a specified body wt than a subsequent wt increment. The opposite set of conditions reduces the probability of neoplasm occurrence, which is even further minimized if the proportion of protein in the diet in early postweaning life and the absolute intake of protein during the early adult period are relatively high. Differences in temporal-specific dietary practices and growth responses accounted for differences in the tissue origin of the tumors. (Author abstract) (44 Refs)

15. MUTAGENICITY STUDY OF FRIED SAUSAGES IN SALMONELLA, DROSOPHILA AND MAMMALIAN CELLS IN VITRO AND IN VIVO.

Gocke E, Eckhardt K, King MT, Wild D
Inst. Molecular Biology, Univ. Aarhus, 8000 Aarhus, Denmark
Mutat Res; 101(4):293-304 1982

An extract of pan-fried sausage was tested in an array of systems, including the Salmonella/microsome test, the Drosophila sex-linked recessive lethal test, tests for induction of sister-chromatid exchange and point mutations in V79 Chinese hamster cells, the micronucleus test, the intrasanguine host-mediated assay, and the mammalian spot test. Results suggest that mice and possibly mammals in general are less sensitive than Salmonella towards the mutagens produced during frying of meat. The mutagenic risk of traces of promutagens in fried meat is orders of magnitude lower than would be estimated using the Ames plate test as a guideline. However, genetic activity of the mutagenic principles of fried meat in mammalian systems cannot be excluded. (29 Refs)

16. INHIBITORY EFFECT OF A FAT-FREE DIET ON MAMMARY CARCINOGENESIS IN RATS.

Davidson MB, Carroll KK
Dept. Biochemistry, Univ. Western Ontario, London, Ontario, N6A 5C1, Canada
Nutr Cancer; 3(4):207-215 1982

Young female Sprague-Dawley rats were administered 7,12-dimethylbenb(a)anthracene, and a wk later the rats were transferred from commerical feed to a semipurified diet containing

20% corn oil. Eight wk after receiving the carcinogen, half of the rats were changed to a fat-free diet to determine the effects on mammary tumor growth and development. After another 20 wk, rats fed the fat-free diet had significantly fewer tumors per tumor-bearing rat and the tumors were smaller than those of rats that continued on the high-fat diet. Rats fed the fat-free diet weighed somewhat less, but showed no physical evidence of essential fatty acid deficiency. Tumors regressed in about half of the rats on the fat-free diet and in some cases became nonpalpable. After 28 wk on this diet, the remaining rats were transferred back to the high-fat diet and subsequently showed a marked stimulation in tumor growth and development. This continued even after the rats were returned to the fat-free diet 8 wk later, indicating that the tumors were no longer susceptible to the deprivation of dietary fat. The results of this study provide further evidence that dietary fat affects the promotional stage of mammary carcinogenesis. (Author abstract) (23 Refs)

17. ISOLATION AND IDENTIFICATION OF KAHWEOL PALMITATE AND CAFESTOL PALMITATE AS ACTIVE CONSTITUENTS OF GREEN COFFEE BEANS THAT ENHANCE GLUTATHIONE (GSH) S-TRANSFERASE ACTIVITY (MEETING ABSTRACT).

Lam LK, Sparnins VL, Wattenberg LW
Department of Laboratory Medicine and Pathology, University of Minnesota, Minneapolis, MN, 55455
Proc Am Assoc Cancer Res; 23:346 1982

GSH S-transferase is a major detoxification system that catalyzes the binding of a variety of electrophiles, including reactive forms of carcinogens, to GSH. Green coffee beans fed in the diet enhance the activity of this enzyme system in the mucosa of the small bowel and the liver of female ICR/Ha mice. Two compounds that produce a marked increase in GSH S-transferase activity were isolated from green coffee beans and identified. They are kahweol palmitate and cafestol palmitate. Both are diterpene esters. Twenty-four hours after 4 daily doses of these two compounds at 5 mg/mouse, GSH S-transferase activity is increased as follows test/controls: kahweol palmitate - liver 3.5, small bowel 5.6; cafestol palmitate - liver 3.0, small bowel 3.9. The free alcohols, ie kahweol and cafestol, and their monoacetates likewise increase GSH S-transferase activity. Palmitic acid does not. In the isolation, the diterpene esters were extracted from green coffee beans into petroleum ether. The petroleum ether extract was fractionated by preparative normal phase, and reverse phase liquid chromatography successively. Final purification with silver nitrate impregnated thin layer chromatography yielded the pure palmitates of cafestol and kahweol. The structures were determined by examination of the spectroscopic data of the esters and their parent alcohols, and by derivative comparison. Several compounds potent in increasing GSH S-transferase activity inhibit carcinogen-induced neoplasia. (no Refs)

18. DIET IN THE EPIDEMIOLOGY OF BREAST CANCER.

Graham S, Marshall J, Mettlin C, Rzepka T, Nemoto T, Byers T
Dept. Social and Preventive Medicine, State Univ. New York at Buffalo, 2211 Main St., Buffalo, NY, 14214
Am J Epidemiol; 116(1):68-75 1982

A variety of studies have shown that diets high in fat, particularly polyunsaturated, have enhanced the production of tumors in animals challenged with chemical carcinogens. Other studies have found an apparent contradiction of no difference in the incidence of breast cancer among women with varying levels of serum cholesterol as measured decades earlier. The present study concerns 2,024 breast cancer cases and 1,463 control patients without neoplasms or pathology of the reproductive and digestive organs, seen at Roswell Park Memorial Institute from 1958 to 1965. Based upon the assessments of their varying ingestion of fats from their own reports of diets, no difference in risk was found. Similarly, there was no difference in risk of breast cancer associated with ingesting diets containing various levels of either vitamin C or the cruciferous vegetables. Risk for breast cancer in women 55 yr of age and older increased somewhat with decreases in ingestion of foods containing vitamin A. (Author abstract) (52 Refs)

19. RELATIONSHIP OF SOYBEAN PASTE SOUP INTAKE TO GASTRIC CANCER RISK.

Hirayama T
Epidemiology Div., Natl. Cancer Center Res. Inst., 1-1, Tsukiji 5-chome, Chuo-ku, Tokyo, 104, Japan
Nutr Cancer; 3(4):223-233 1982

Daily intake of soybean paste soup was found to significantly reduce standardized mortality rates for gastric cancer in an ongoing large-scale prospective study of 122,261 males and 142,857 females aged 40 and above in 29 Japanese health center districts, 1966-1978. The gastric cancer standardized mortality rates were 171.9, 210.2, 240.0, and 255.9 per 100,000 males, and 77.8, 85.3, 97.5, and 113.6 per 100,000 females in daily, occasional, rare, and noningesters, respectively. These relationships remained significant when observed by age group, district, socioeconomic status, dietary pattern, and smoking habit. The risk-reducing effect has also been observed in case-control studies in the past, for both males and females and in urban and rural areas. This beneficial effect could arise from selected compounds such as protease inhibitors and/or other nutritious substances included in the soybean, but it is also possible that it merely reflects the effect of some frequent accompaniment to soybean paste soup, such as green-yellow vegetables. (Author abstract) (16 Refs)

20. BREAST TUMORS INDUCED BY N-NITROSOMETHYLUREA (NMU) OR DIMETHYLBENZ(A)ANTHRACENE (DMBA) IN CASTRATED MALES: ROLE OF HIGH FAT DIET (MEETING ABSTRACT).

Rivenson A, Silverman J, Weisburger J
American Health Foundation, Valhalla, NY, 10595
Proc Am Assoc Cancer Res; 23:275 1982

We attempted to extend the findings of Fingerhut and Veenema on the induction of prostate cancer in gonadectomized male Fischer F-344 rats on 20% and 5% fat diets, utilizing iv dosing with 7,12-dimethylbenz(a)anthracene (DMBA), and also 2',3-dimethyl-4-aminobiphenyl (DMAB), and N-nitrosomethylurea (NMU). No prostate cancers were found in rats held for 68 weeks. However, with NMU and DMAB these gonadectomized male rats had breast cancers in a higher incidence on the 20% fat diet with NMU but not with DMAB. All neoplasms were malignant, with invasion and destruction of neighboring tissues, and presenting histologically mixed glandular and squamous structures, quite distinct from lesions usually seen in females. Similar type of adeno-squamous carcinomas could be seen in tumors of the ear duct (Zymbal glands) and bulbo urethral glands developed in rats given the above carcinogens. The topographic location of the tumors labeled grossly as 'breast tumors' excluded other possible similar glandular structures and also a sweat gland tumor since the rat skin does not contain sweat glands. Other tumors noted after the carcinogens

given iv were: bladder, DMAB only; small intestine and colon, NMU and DMAB; lung, DMBA, NMU, and DMAB; sc sarcoma, NMU and DMAB; stomach NMU. Several of these cancers exhibited metastases. None of the tumors noted above were present in controls. In summary, NMU and DMAB induced tumors in a number of sites, but selectively caused mammary gland adenocarcinoma and adenosquamous carcinoma, and Zymbal gland adenoepidermoid carcinoma. (1 Ref)

21. EFFECT OF DIETARY FAT AND ANTIOXIDANT ON INCIDENCE OF SPONTANEOUS AND 1,2-DIMETHYLHYDRAZINE-INDUCED TUMORS IN MICE (MEETING ABSTRACT).

Brown RR, Erturk E, Arend RA, Potera C, Remus N, Ruzicka E, Dallmann L

University of Wisconsin Center for Health Sciences, Madison, WI, 53792

Proc Am Assoc Cancer Res; 23:277 1982

In attempts to further define the role of type and quantity of dietary fat and antioxidants on cancer incidence, we studied tumor incidence (spontaneous and dimethylhydrazine (DMH) induced) in mice fed semipurified diets having one of 5 different vegetable fat mixes containing respectively 59% saturated (SAT), 60% monoene (MUF), 59% diene (DUF), 35% transmonoene (TRN), or 4% triene (TUF). Butylatedhydroxyanisole (BHA) was added to some SAT and TUF diets. Weanling CD-1 mice of both sexes (40/group) and female C3H (60/group) were fed these diets having 5% or 17% fat substituted isocalorically for sucrose. Treated mice received 10 weekly subcutaneous injections of 0.25 mg DMH freshly neutralized. Controls received buffer only. DMH caused liver tumors and a few kidney and lung tumors and splenic leukemia, but no colon tumors. More DMH-induced liver and lung tumors were seen in males fed 5% TRN than in other groups but this was not confirmed in males fed 17% TRN. Total tumors in males fed 17% and 5% fats were not different suggesting that when fed isocalorically, the quantity of fat did not influence tumor incidence in CD-1 males. BHA reduced the DMH-liver tumor rate in 17% SAT but not in 17% TUF. DMH-liver tumors were fewer in 17% SAT than in 17% TUF males and this difference was even greater between SAT+BHA+DMH and TUF+BHA+DMH groups. (no Refs)

GENERAL REVIEWS AND OTHER STUDIES

22. THE NITROSAMINE HAZARD.

Yonkers AJ

Dept. Otolaryngology, Univ. Nebraska, Omaha, NE

Ear Nose Throat J; 61(7):407-414 1982

Experimental, clinical, and biochemical studies of the possible hazard of nitrosamines in the environment to human populations are reviewed. Nitrosamines are also discussed in relation to occurrence in various foods, possible relationship to cancers in certain geographic, their role as adjuvants, and the function of some blocking agents. (65 Refs)

23. INFLUENCE OF NITRATES ON THE HEALTH STATUS OF THE POPULATION.

Vondrakova M

Institut hygieny a epidemiologie, Srobarova 48, 100 42 Prague 10, Czechoslovakia

Cesk Hyg; 27(3):162-172 1982

Studies on the health effects of nitrates and nitrites are reviewed. Nitrates and nitrites possibly participate in the formation of carcinogenic N-nitroso compounds (N-nitrosodimethylamine, N-nitrosodiethylamine, N-nitrosopyrrolidone and N-nitrosopiperidine) in foods and organisms. Recent epidemiological studies show a correlation between the incidence of carcinoma of certain localizations (especially of the stomach) and the presence of nitrosamines, nitrates and nitrites in the environment. (80 Refs) (In CZE)

24. ZINC, COPPER, AND MANGANESE IN IMMUNE FUNCTION AND EXPERIMENTAL ONCOGENESIS.

Beach RS, Gershwin ME, Hurley LS

Dept. Internal Medicine, Div. Rheumatology-Clinical Immunology, Univ. Calif., Davis, CA, 95616

Nutr Cancer; 3(3):172-191 1982

Dietary deficiency of trace metals among human populations, once thought to be exceedingly rare, has recently gained attention as a potential public health problem. The consumption of highly refined and heavily processed foods reduces the trace element content of the diet. The higher trace element requirements of pregnancy, lactation, growth, development, and chronic disease may further contribute to state of marginal trace metal nutriture. Experimental animal studies have demonstrated that even marginal trace element deprivation during critical periods of growth and development or, alternately, during prolonged deficiency in adults can significantly alter immunologic function. Furthermore, trace metal nutriture has been shown to affect initiation and progression of a large variety of neoplasia. Studies of the interaction of trace metal nutriture and cancer have, however, suffered from many methodological inconsistencies as well as failures to define and quantitate the trace element content of diets and the host alterations in response to neoplastic challenge. Similarly, there has been little information in the critical area of marginal and moderate trace metal deficiency, i.e., those experimental situations most analogous to deficiencies that may occur in human populations. In this review, an attempt is made first to place in perspective experimental data relevant to these issues, and second to emphasize the desirability of further investigation in this critical area of human nutrition. (Author abstract) (186 Refs)

25. CARCINOGENESIS AND BIOLOGICAL EFFECTS OF TUMOR PROMOTERS.

Hecker E

Inst. Biochemistry, German Cancer Res. Center, Heidelberg, W. Germany

Carcinogenesis 7: 664 pp., 1982.

The most recent experimental findings on the etiological, cell biological, and molecular facets of carcinogenesis are reviewed. The basic problems of whether specific carcinogens function as promoters of carcinogenesis, as stimulators of initiation, or both, are addressed with respect to in vivo animal studies, epidemiological studies, and in vitro studies of tumor promotion. Carcinogenesis and tumor promotion in vivo is discussed with respect to the skin as a target tissue and nonepidermal target tissues. Effects of tumor promoters on cell proliferation and differentiation are reviewed for the epidermis and

nonepidermal cell systems. Membrane interactions and receptors, effects on intercellular communication and cell metabolism, and viral and immunologic aspects are reviewed. Epidemiologic studies of esophageal cancer in Linxian County, China, nutritional carcinogenesis, and breast cancer as an example of promoting effects are discussed. (1368 Refs)

26. INHIBITION OF TUMOR INDUCTION AND DEVELOPMENT.

Zedeck MS, Lipkin M
Memorial Sloan-Kettering Cancer Center, New York, NY
Inhibition of Tumor Induction and Development. New York, Plenum Press, 233 pp., 1981.

Major aspects of chemoprevention, focusing on the inhibition of carcinogen formation, absorption, and metabolic activation and interaction with cell macromolecules, as well as prevention through immunological approaches, are outlined. Inhibition of chemical carcinogenesis by phenols, coumarins, aromatic isothiocyanates, flavones, and indoles and inhibition of carcinogen metabolisms and action by disulfiram, pyrazole, and related compounds are discussed. Retinoids and chemoprevention of cancer; ascorbic acid inhibition of N-nitroso compound formation in chemical, food, and biological systems; alpha-tocopherol and its relationship to tumor induction and development; trace elements and metals as anticarcinogens; and the protective role of plant sterols in chemical carcinogenesis are described. Immunoprevention is discussed with respect to detection of tumor-associated antigens in experimental rat bowel carcinomas, evidence that embryonic antigens are associated with bowel carcinomas, enhanced 1,2-dimethylhydrazine (DMH)-induced tumorigenesis in imunosuppressed rats, inhibitory effect on bowel carcinogenesis by immunization with transplantable syngeneic colon carcinoma and by immunization with fetal tissue, inhibition of DMH-induced carcinogenesis in multiparous rats, effects of tumor resection on the development of additional primary tumors, and regression of early primary bowel carcinomas by multimodal immunological treatment are discussed. (738 Refs)

27. SECOND ANNUAL REPORT ON CARCINOGENS.

National Toxicology Program
(S. d'Arazien) Public Information Office, Natl. Toxicology Program, MD B2-04, P.O. Box 12233, Research Triangle Park, NC, 27709
Second Annual Report on Carcinogens. Washington, D. C., National Toxicology Program, 447 pp., 1981.

Information on the nature and degree of exposures to substances which are either known to be carcinogens and to which a significant number of persons residing in the United States are exposed and the extent to which Federal regulations are effective in reducing the risk to the public health from exposures to these chemicals is presented. Substances to which only a few people are exposed, for the most part are not included. Some chemicals have been included that have been banned or whose use has been restricted because people previously exposed who are potentially at risk are still living, or because some of these chemicals still remain in the environment. Eighty-eight chemicals, including 25/26 from the First Annual Report are presented; chloramphenicol has been dropped because strength of evidence of human data is inadequate and no data on experimental animals exist. Each chemical is described, including a brief synopsis of the scientific evidence concerning carcinogenicity. Two of the chemicals, aflatoxin and cycasin, occur naturally. Two food or cosmetic additives with the potential for major use and exposure are saccharin and safrole. Fourteen pharmaceuticals include six anticancer drugs. Eight pesticides have been included. The other chemicals are industrial chemicals, miscellaneous chemicals and analogs, or industrial processes or byproducts. Appendixes of individual chemicals and their synonyms, acronyms, and measurements are presented. (no Refs)

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. ASBESTOS CARCINOGENESIS: ASBESTOS INTERACTIONS AND EPITHELIAL LESIONS IN CULTURED HUMAN TRACHEOBRONCHIAL TISSUES AND CELLS.
Haugen A, Harris C
Div. Cancer Cause and Prevention, NCI, Bethesda, MD
Recent Results Cancer Res; 82:32-42 1982

2. REVIEW: ORAL CONTRACEPTIVES AND MENOPAUSAL ESTROGENS IN RELATION TO BREAST NEOPLASIA.
Brzezinski A, Schenker JG
Dept. Obstetrics and Gynecology, Hadassah Univ. Hosp., POB 12000, 91120 Jerusalem, Israel
Isr J Med Sci; 18(4):433-438 1982

3. LIVER DISEASES DUE TO HORMONAL CONTRACEPTIVES.
Lie TS
Chirurgische Klinik und Poliklinik, Universitat, Sigmund-Freud-Str. 25, D-5300 Bonn 1, W. Germany
MMW; 124(20):489-494 1982
In GER

RADIATION CARCINOGENESIS

4. THE WORK OF THE NRPB 1977/80 AND A REVIEW OF THE FIRST 10 YEARS.
National Radiological Protection Board
Natl. Radiological Protection Board, Chilton, Didcot, Oxfordshire OX11 0RQ, England
The Work of the NRPB 1977/80 and a Review of the First 10 Years. Chilton, Didcot, Oxfordshire, National Radiological Protection Board, 70 pp., 1981.

DIETARY FACTORS IN CARCINOGENESIS

5. ALCOHOL AND CARCINOGENESIS.
Seitz HK, Czygan P, Kommerell B
Schwerpunkt Gastroenterologie, Medizinische Universitatsklinik, Bergheimer Str. 58, D-6900 Heidelberg 1, W. Germany
Leber Magen Darm; 12(3):95-107 1982
In GER

6. ZINC, COPPER, AND MANGANESE IN IMMUNE FUNCTION AND EXPERIMENTAL ONCOGENESIS.
Beach RS, Gershwin ME, Hurley LS
Dept. Internal Medicine, Div. Rheumatology-Clinical Immunology, Univ. Calif., Davis, CA, 95616
Nutr Cancer; 3(3):172-191 1982

EPIDEMIOLOGY, ETIOLOGY AND GENETIC ASPECTS OF HUMAN CANCER

7. TRENDS IN CANCER INCIDENCE: CAUSES AND PRACTICAL IMPLICATIONS. PROCEEDINGS OF A SYMPOSIUM HELD OSLO, NORWAY, 6-7 AUGUST 1980.
Magnus K
The Cancer Registry Norway, Montebello, Oslo 3, Norway
Trends in Cancer Incidence: Causes and Practical Implications, Proceedings of a Symposium held Oslo, Norway, 6-7 August 1980. Washington, D. C., Hemisphere Publishing Corporation, 446 pp., 1982.

ENVIRONMENTAL AND OCCUPATIONAL FACTORS IN CARCINOGENESIS

8. QUANTIFICATION OF OCCUPATIONAL CANCER.
Peto R, Schneiderman M
Nuffield Dept. Clinical Medicine, Radcliffe Infirmary, Univ. Oxford, Oxford, England
Banbury Rep (9): 756 pp., 1981.

9. NASAL CARCINOMA IN WOODWORKERS: A REVIEW.
Wills JH
Srh. Medicine, Uniformed Services Univ. Health Sciences, 4301 Jones Bridge Road, Bethesda, MD, 20814
JOM; 24(7):526-530 1982

10. AIR POLLUTION AND CANCER. A REVIEW OF EXPERIMENTAL INVESTIGATIONS.
Madsen ES, Nielsen PA, Pedersen JC
(c/o Nielsen), Stigardsvej 2, DK-2900 Hellerup, Denmark
Ugeskr Laeger; 144(23):1719-1721 1982
In DAN

11. AIR POLLUTION AS A RISK FACTOR IN LUNG CANCER.
Vena JE
Dept. Social and Preventive Medicine, State Univ. New York at Buffalo, 2211 Main St., Buffalo, NY, 14214
Am J Epidemiol; 116(1):42-56 1982

12. INHALATION CARCINOGENESIS: AN OVERVIEW.
Chretien J
Hopital Laennec, 42 rue du Sevres, F-75007 Paris, France
Recent Results Cancer Res; 82:1-10 1982

13. THE NITROSAMINE HAZARD.
Yonkers AJ
Dept. Otolaryngology, Univ. Nebraska, Omaha, NE
Ear Nose Throat J; 61(7):407-414 1982

CELLULAR AND MOLECULAR MECHANISMS IN CARCINOGENESIS

14. ONCOGENES OF SPONTANEOUS AND CHEMICALLY INDUCED TUMORS.
Weinberg RA
Center for Cancer Res., Massachusetts Inst. Technology, Cambridge, MA, 02139
Adv Cancer Res; 36:149-163 1982

15. RELATIONSHIP OF DNA TERTIARY AND QUATERNARY STRUCTURE TO CARCINOGENIC PROCESSES.
Lipetz PD, Galsky AG, Stephens RE
Dept. Radiology, Ohio State Univ., Columbus, OH, 43210
Adv Cancer Res; 36:165-210 1982

16. ROLE OF ENZYMATIC METHYLATION OF DNA IN CELL DIFFERENTIATION AND TRANSFORMATION.
Fedorov NA
Central Res. Inst. Hematology and Blood Transfusion, Moscow, USSR
Eksp Onkol; 4(3):3-11 1982
In RUS

17. HYDROGEN ION DYNAMICS AND CANCER: AN APPRAISAL.
Harguindey S
Dept. Thoracic Surgery and Oncology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263
Med Pediatr Oncol; 10(3):217-236 1982

MODIFICATION OF CARCINOGENESIS

18. CARCINOGENESIS AND BIOLOGICAL EFFECTS OF TUMOR PROMOTERS.
Hecker E
Inst. Biochemistry, German Cancer Res. Center, Heidelberg, W. Germany
Carcinogenesis 7: 664 pp., 1982.

VIRAL AND IMMUNOLOGICAL ASPECTS OF CARCINOGENESIS

19. AVIAN LEUKEMIA VIRUSES: ONCOGENESES AND GENOME STRUCTURE.
Graf T, Stehelin D
Inst. Virology, German Cancer Res. Center, Im Neuenheimer Feld 280, 6900 Heidelberg, W. Germany
Biochim Biophys Acta; 651(4):245-271 1982

20. HYPERPLASTIC AND NEOPLASTIC ERYTHROPROLIFERATIVE DISEASES INDUCED BY ONCOGENIC MURINE RETROVIRUSES.
Scolnick EM
Lab. Tumor Virus Genetics, NCI, Bethesda, MD, 20205
Biochim Biophys Acta; 651(4):273-283 1982

METHODOLOGY IN CARCINOGENESIS RESEARCH

21. MOLECULAR EPIDEMIOLOGY AND CARCINOGEN-DNA ADDUCT DETECTION: NEW APPROACHES TO STUDIES OF HUMAN CANCER CAUSATION.
Perera FP, Weinstein IB
Div. Environmental Sciences, Columbia Univ., New York, NY, 10032
J Chronic Dis; 35(7):581-600 1982

22. AN EVALUATION OF ASSAY PROCEDURES FOR DETECTION OF TUMOR PROMOTORS.
Sivak A
(c/o P. H. Lohman) Medical Biological Lab. TNO, P.O. Box 45, 2280 AA Rijswijk, The Netherlands
Mutat Res; 98(3):377-387 1982

23. DNA REPAIR ASSAYS AS TESTS FOR ENVIRONMENTAL MUTAGENS. A REPORT OF THE U.S. EPA GENE-TOX PROGRAM.
Larsen KH, Brash D, Cleaver JE, Hart RW, Maher VM, Painter RB, Sega GA
Environmental Mutagen Information Center, Oak Ridge Natl. Lab., Oak Ridge, TN, 37830
Mutat Res; 98(3):287-318 1982

24. AN EVALUATION OF THE HOST-MEDIATED ASSAY AND BODY FLUID ANALYSIS. A REPORT OF THE U.S. ENVIRONMENTAL PROTECTION AGENCY GENE-TOX PROGRAM.
Legator MS, Bueding E, Batzinger R, Connor TH, Eisenstadt E, Farrow MG, Ficsor G, Hsie A, Seed J, Stafford RS
Dept. Preventive Medicine and Community Health, Div. Environmental Toxicology, Univ. Texas Medical Branch, Galveston, TX, 77550
Mutat Res; 98(3):319-374 1982

25. THE PHYSIOLOGICAL IMPORTANCE OF THE COLONIC MICROFLORA.
Gustafsson BE
Dept. Germfree Res., Karolinska Inst., Box 60 400, S-104 01 Stockholm, Sweden
Scand J Gastroenterol [Suppl]; 17(77):117-131 1982

26. AN ASSESSMENT OF THE ROLE OF MICRODOSIMETRY IN RADIOBIOLOGY.
Goodhead DT
Medical Res. Council Radiobiology Unit, Harwell, Didcot OX11 ORD, England
Radiat Res; 91(1):45-76 1982

OTHER REVIEWS IN CARCINOGENESIS

27. PSYCHOSOCIAL ASPECTS OF CANCER.
Cohen J, Cullen JW, Martin LR
Sch. Social Welfare, Univ. California, Los Angeles, Los Angeles, CA
Psychosocial Aspects of Cancer. New York, Raven Press, 320 pp., 1982.

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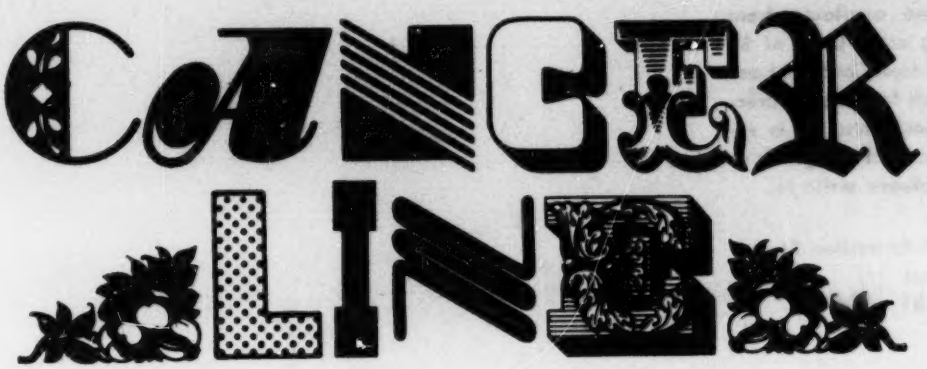

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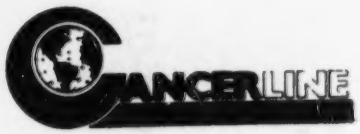

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DIETARY ASPECTS OF CARCINOGENESIS

SERIES CK03 No. 82/12

DECEMBER, 1982

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- **MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS..... 7-14**
- **GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS..... 15-28**
- **GENERAL REVIEWS AND OTHER STUDIES..... 29**

STUDIES OF NATURALLY OCCURRING CARCINOGENS IN THE DIET

1. FORMATION OF MUTAGENS IN COOKED FOODS. V. THE MUTAGEN REDUCING EFFECT OF SOY PROTEIN CONCENTRATES AND ANTIOXIDANTS DURING FRYING OF BEEF.

Wang YY, Vuolo LL, Spingarn NE, Weisburger JH
(c/o Weisburger) Naylor Dana Inst. for Disease Prevention, American Health Foundation, Valhalla, NY, 10595
Cancer Lett; 16(2):179-189 1982

The mutagenicity of fried beef patties with various amounts of added soy protein concentrates (SPC) was studied using the Ames Salmonella/mammalian enzymes assay. Beef-SPC patties are quite common and have been used in school lunch programs in the United States. The optimum amount of S-9 required for fried beef mutagens was 50 ul of S-9 in 0.5 ml of S-9 mix/plate. The protein concentration of the S-9 was 29 mg/ml. The mutagenicity of beef decreased as the percentage of added SPC increased. The composition of SPC used was 71.5% protein, 17.7% carbohydrates, 0.3% fat, and 3.5% crude fiber and minor components. The patties were cooked under controlled,

HE20.3173/2:

reproducible conditions for 5 min/side producing medium-done patties. At 4% of added SPC, soy-hamburgers exhibited less mutagenicity than regular hamburgers cooked for a shorter time. The mutagen reducing action of SPC occurred during the cooking process since mixing extracts of separately cooked beef patties and SPC did not reduce the mutagenicity. The addition of SPC did not affect the action of the activating enzymes in S-9 fraction. With beef plus gluten, the mutagenicity was reduced, but to a some what lesser degree than SPC. Casein plus beef did not reduce the mutagenicity, indicating that protein alone is not responsible for the reduction. The mutagen reducing ability of SPC may be due to both volumetric and chemical effects. The phenolic antioxidant components of SPC may also play a role. (29 Refs)

STUDIES OF SYNTHETIC ADDITIVES OR CONTAMINANTS OF THE DIET

2. DOSE-RESPONSE STUDIES ON THE INHIBITION OF EXPERIMENTAL MAMMARY, COLON, AND LIVER CARCINOGENESIS BY BUTYLATED HYDROXYTOLUENE (BHT) (MEETING ABSTRACT).

Cohen LA, Reddy BS, Williams GM, Furuya K, Maeura Y, Silverman J, Weisburger JH
Naylor Dana Institute, American Health Foundation, Valhalla, NY, 10595
Proc Am Assoc Cancer Res; 23:358 1982

Phenolic antioxidants such as BHT inhibit carcinogenesis at several target organs including breast, colon, liver, lung, and stomach. We investigated (1) whether a dose response relationship exists between the tumor inhibiting effect and BHT level, and (2) whether the inhibiting is influenced by the dose of initiating carcinogen in three well-characterized model systems: the dimethyl-benz(a)anthracene (DMBA)-induced mammary carcinoma, the azoxymethane-induced colon carcinoma, and the 2-acetylaminofluorene (2-AAF)-induced hepatocarcinoma. Two appropriate doses of initiating carcinogen were used and BHT was fed in the diet at concentrations of 6000, 3000, 1000, and 300 ppm. In mammary carcinogenesis, tumor incidence was 75% and 55% and tumor multiplicity 2.5 and 1, respectively, at high (15 mg) and low (5 mg total) dose DMBA. With the high DMBA dose, tumor incidence and multiplicity were significantly inhibited only at the highest level of BHT. At low DMBA dose, tumor incidence and multiplicity were inhibited at all levels of BHT. In colon carcinogenesis, at the high dose of azoxymethane, inhibition as determined by endoscopy was evident at the two higher doses of BHT. In the liver model, 6 weeks of feeding 0.02% AAF produced 26 preneoplastic foci/cm2 liver, identified by resistance to iron accumulation. Concurrent feeding of BHT produced a dose-dependent inhibition of foci production. Thus, BHT exerts a dose-dependent inhibition of carcinogenesis in all three model systems. (no Refs)

3. N-NITROSOPROLINE CONCENTRATION IN FOOD PRODUCTS.

Lettner AKh, Kann IuM, Tauts OV
Depth Food Technology, Estonian Polytechnical Inst., Tallin, USSR
Vopr Pitan; (3):64 1982

Concentration of N-nitrosoproline was determined in various food products. Highest levels were observed in smoked meat products (90-151 ug/kg) and in smoked cheese (130 ug/kg). The level in smoked fish was 17 ug/kg. Findings of the study indicated that high temperatures enhance the nitrosation. (2 Refs) (In RUS)

4. INVESTIGATION, WITH THE SALMONELLA/MICROSOME TEST, OF PSYCHOPHARMACEUTICALS USED IN MEAT PRODUCTION.

Preiss AM, Scheutwinkel-Reich M, Fulle I, Grohmann HG, Stan HJ
Institut fur Lebensmittelchemie, Technische Universitat Berlin, Muller-Breslau-Strasse 10, 1000 Berlin 12, W. Germany
Mutat Res; 104(6):333-337 1982

Mutagenic activity of four veterinary drugs, azaperone, acepromazine, xylazine, and diazepam, was investigated in the Salmonella/microsome test using five histidine-auxotrophic strains, with and without metabolic activation. These compounds are used to reduce aggressiveness and activity during live-stock breeding, and their residues may be found in meat consumed by humans. Only azaperone and xylazine were weakly mutagenic and further studies of these drugs is recommended. (8 Refs)

5. MYCOTOXINS AS A HUMAN HEALTH HAZARD.

Ghosh AC
Sisa, Inc., Cambridge, MA
Mycotoxins as a Human Health Hazard. Available from National Technical Information Service, Springfield, VA, as PB82-191057, 23 pp., 1981.

The development of a methodology to detect Penicillium islandicum Sopp and other penicillia toxins in various food grains and food stuffs is described. The microbial production of luteoskyrin and other metabolites of P islandicum is discussed and the development of detection techniques for toxins elaborated by P islandicum is described. These techniques include thin layer chromatography cum negative chemical ionization mass spectrometry, high pressure liquid chromatography, and thin layer chromatography for detection of cyclochlorotine and simatoxin. Results are presented of biological studies of antimicrobial activity, antitumor activity, and mutagenicity. It was found that luteoskyrin is an effective antimicrobial agent against a number of gram-positive and gram-negative organisms. Luteoskyrin, determined to be a non-mutagenic against various strains of Salmonella typhimurium, was also found to possess antitumor activity against P388 lymphocytic leukemia. The identification of cyclochlorotine through mass spectrometric peptide sequencing is noted. (Author abstract)

6. DIFFERENTIAL EFFECTS OF DIETARY AND INTRAPERITONEAL ADMINISTRATION OF ANTIOXIDANTS ON THE ACTIVITIES OF SEVERAL HEPATIC ENZYMES OF MICE.

Cha YN, Heine HS, Moldeus P
Dept. Pathobiology, Johns Hopkins Medical Institutions, Baltimore, MD
Drug Metab Dispos; 10(4):434-435 1982

Biochemical effects of ip administration 2(3)-tert-butyl-4-hydroxyanisole (BHA), 1,2-dihydro-6-ethyl-2,2,4-trimethylquinoline (EQ), and disulfiram (DSF) on female CD-1 mice were compared with those previously observed by dietary administration of the antioxidants. Dietary administration of BHA and EQ produced marked enlargement of the liver. In contrast, ip administration of the antioxidants for the same duration did not alter the liver weights. Pretreatment of mice with DSF, either in the diet or by ip injections, did not produce any changes in their liver weights. Feeding of BHA, EQ, and DSF all pro-

duced disproportionate increases in the activity of both flavin reductases. BHA produced a moderate increase of NADPH-cytochrome c reductase activity without any changes in cytochrome P450 levels. No effects on aminopyrine N-demethylation were noted but a marked increase of p-hydroxylation of aniline and a significant decrease of benzopyrene mono-oxidation occurred. Dietary EQ produced an increase in NADPH-cytochrome c reductase activity and marked elevation of cytochrome P450 levels. Concurrent with the increases, the rates of N-demethylation and of hydroxylation were greatly increased. However, the oxidation rate of benzopyrene was reduced. Changes produced by DSF were small if any. Differential effects produced on glucose 6-phosphate, glutathione reductase, and uridine diphosphate-glucose dehydrogenase are described. (17 Refs)

MODIFICATION OF CARCINOGENESIS BY ESSENTIAL NUTRIENTS

7. DIETARY PROTEIN CONCENTRATION AND THE METABOLISM OF 1,2-DIMETHYLHYDRAZINE BY MICE.

Kari FW
University of Illinois at Urbana-Champaign, Urbana, IL
Diss Abstr Int [B]; 42(9):3636-B 1982

At concentrations ranging from 2.5 to 10 mM, 1,2-dimethylhydrazine (DMH) and azoxymethane (AOM) did not increase the mutation frequency of Salmonella typhimurium (Strain G-46) in vitro. Murine liver microsomes and cofactors caused a 40-fold increase in mutation frequency with dimethylnitrosamine (DMN) but not with DMH or AOM in vitro. Methylazoxymethanol (MAM) was mutagenic in vitro without activation by liver preparations. The conversion of DMH, AOM, and MAM to mutagens was studied in growing male B6C3F1 mice fed isocaloric, semipurified casein, sucrose:dextrin diets containing 2.5, 5, 10, 20, or 40% protein. Mutagen formation from DMH and MAM varied slightly with diet but from AOM was directly proportional to dietary protein concentration. The percent of [14C]-DMH expired as a promutagen by growing mice fed 2.5, 10, or 40% protein was 63 +/-4, 44 +/-3, and 38 +/-3. The metabolism of DMH to mutagenic endproducts by mice was rate-limited by the conversion of azomethane to AOM and at least partially carried out by the liver. Increasing protein intake increased the bioactivation of DMH, AOM, and MAM and 2.5% protein diets caused greater expiration of promutagens from DMH, thereby decreasing the body burden of retained carcinogen. (no Refs)

8. EFFECT OF INCREASING LEVELS OF DIETARY PROTEIN ON THE METABOLIC ACTIVATION AND COVALENT BINDING OF AFLATOXIN B1 (MEETING ABSTRACT).

Hayes JR, McCall CO, Bagley DM
Department of Pharmacology Medical College of Virginia, Virginia Commonwealth University, Richmond, VA
Toxicologist; 2(1):25 1982

Low dietary protein levels (5%) decrease the hepatocarcinogenicity of aflatoxin B1 (AFB1), in part due to decrease in hepatic microsomal mixed function oxidase (MFO) activity, which activates AFB1 to its ultimate carcinogen. Although low levels of dietary protein are consumed by certain populations, others may consume protein above that required for maintenance. This study was designed to ascertain the effects of increasing levels of dietary protein on the metabolic activation of AFB1. Male, weanling Sprague-Dawley derived rats fed the AIN-76 semi-purified diet with casein levels of 5, 10, 20, 30 and 40% for 10 days. Body weight increased until protein levels reached 20% and then became asymptotic. Liver weights were higher only in the 5 and 10% groups due to lipid infiltration. Microsomal protein increased with dietary protein up to 20% and then became asymptotic. N-demethylation of ethyl-morphine (EM) increased until dietary protein levels reached 30% and then became constant. Cytochrome c reductase activity and cytochrome P-450 levels followed a trend identical to EM metabolism. In vitro metabolic activation of AFB1 to a produce(s) capable of covalently binding DNA increased until a level of 30% protein was reached and then became asymptotic. In vivo covalent binding of an AFB1 metabolite(s) to hepatic DNA, RNA and protein followed the same trend as the in vitro studies. Inappropriately low levels of dietary protein may be protective in respect to AFB1 carcinogenicity, but high levels may increase risk. (no Refs)

9. EFFECT OF DIETARY FAT TYPE ON THE TOXICITY, METABOLISM AND MUTAGENICITY OF N-NITROSODIMETHYLAMINE IN MICE (MEETING ABSTRACT).

Bunce OR, Evans B, Walton L, Rodeheaver D, Wade AE
Department of Pharmacology, University of Georgia School of Pharmacy, Athens, GA, 30602
Fed Proc; 41(3):1548 1982

The influence of dietary fat on toxicity, metabolism and mutagenicity of N-nitrosodimethylamine (DMN) was studied in male Swiss Webster mice. Mice fed fat free, tallow (10%), corn oil (10%) and lab chow diets for four weeks were injected with 7 mg/kg, ip of DMN followed by a regimen of 10 PPM in drinking water 5 days/week for 4 weeks. Treatment with DMN decreased serum protein levels and body weights in all mice while significantly increasing serum alkaline phosphatase and glutamic-oxaloacetate transaminase only in corn oil dieted mice. Pathological examination showed no abnormalities in control animals while DMN treatment produced diet related vascular lesions in the central veins and mild ascites due to hypoproteinemia. The catalytic coefficients (Vmax/Km (diet treated)/Vmax/Km FF diet) for demethylase I of post mitochondrial fraction (S-9) of livers from mice fed tallow, corn oil and lab chow diets were increased, while the catalytic coefficients for Demethylase II were decreased in all animals when compared to those fed fat-free diet. DMN treatment reduced the metabolism of DMN which was reflected in a decrease in mutagen formation by S-9 fractions. (no Refs)

10. ZINC DEFICIENCY, METHYL BENZYL NITROSAMINE CARCINOGENESIS AND DNA SYNTHESIS IN ESOPHAGEAL MUCOSA OF THE RAT (MEETING ABSTRACT).

Schrager T, Newberne PM
Department of Nutrition and Federal Science, M.I.T., Cambridge, MA
Toxicologist; 2(1):105 1982

Zinc deficiency causes hyperplasia and parakeratosis in the esophageal epithelium of many animal species, and increases susceptibility of this tissue to chemical carcinogens. Male Sprague-Dawley rats were given either a control diet (60 ppm) or a diet deficient in Zn (3 ppm). After one month on these diets, some rats received 2.5 mg/kg body weight methyl-benzyl nitrosamine (MBN), twice a week for three weeks. At the end of

the dosing period, one group of control ad libitum animals was switched to zinc deficient diet and one group of zinc deficient was switched to control diet. Zinc deficiency throughout the experiment significantly enhanced tumor incidence. Switching diets gave equivocal results. After one month on diets the zinc deficient diet only, led to much greater DNA synthetic activity than the control diet. Following the first dose of MBN, DNA synthetic activity dropped to background levels for both groups but exceeded background at end of dosing. Ten days after the last dose of MBN, DNA synthesis was significantly greater in the control groups. (no Refs)

11. 7,12-DIMETHYLBENZ-(ALPHA)ANTHRACENE (DMBA) CARCINOGENICITY: MODERATION BY SUPPLEMENTAL BETA CAROTENE (B-CAR) AND VITAMIN A (VIT A) (MEETING ABSTRACT).

Rettura G, Levenson SM, Seifter E
Albert Einstein College of Medicine, Yeshiva University, Bronx, NY, 10461
Fed Proc; 41(3):67 1982

Because B-Car inhibits tumor development due to Moloney Sarcoma Virus and C3HBA adenocarcinoma, there is interest in the possible prevention of other tumors by B-Car. Previously we showed that B-Car, given 2 days before DMBA, inhibited DMBA carcinogenesis. We report now the effect of B-Car and Vit A supplementation, started either 1 or 2 weeks after DMBA. One hundred female SD (Sprague-Dawley) rats (180 g) maintained on Purina Lab chow (Vit A + B-Car content - 18,000 units Vit A/Kg diet) received 20 mg DMBA in 1 ml corn oil by stomach tube. Treatments were begun one week later: a) no treatment, b) Vit A supplementation (150,000 units/Kg diet), c) B-Car supplementation (90 mg/Kg diet). One additional group, d), was started on supplemental Vit A the second week after DMBA and one, e), on supplemental B-Car at this time. The results after 22 weeks based on groups of 20 rats each: deaths -4 in group a; rats with tumors -15 in group a, 3 in b, 4 in c, 3 in d, and 4 in e; and tumors/group -30 in group a, 3 in b, 5 in c, 5 in d, and 6 in e. We conclude: B-Car and Vit A are active in preventing DMBA tumors, even when supplementation is started two weeks after DMBA administration. (no Refs)

12. THE EFFECTS OF RIBOFLAVIN DEPLETION AND 3-METHYLCHOLANTHRENE TREATMENT ON HEPATIC AZOREDUCTASE AND D.T. DIAPHORASE (MEETING ABSTRACT).

Newton-Nash DK, Sadowski JA
Marquette University School of Dentistry, Milwaukee, WI, 53233
Fed Proc; 41(3):357 1982

Hepatic azoreductase and DT diaphorase are involved in the metabolism of xenobiotics and carcinogens. Both enzymes are flavoproteins and induced by 3-methylcholanthrene (3MCA). The effect of riboflavin deficiency on hepatic levels of these enzymes and their inductions by 3MCA was examined in male Sprague-Dawley rats fed a deficient diet for 6 weeks. Controls received deficient diet supplemented with riboflavin in the drinking water (20 ug/ml). 3MCA was injected ip daily for three days prior to sacrifice (10 mg/kg). Both enzymes were predominantly located in the cytosol where riboflavin deficiency resulted in a 41.9% decrease in azoreductase and a 59.6% decrease in diaphorase. Microsomal diaphorase and azoreductase were only slightly decreased. 3MCA resulted in a 10 and 14 fold increase in cytosolic azoreductase and diaphorase in controls, whereas deficient rats elicited a 13 and 20 fold increase in the activity of these enzymes. Both cytosolic enzymes were inhibited 60% by Warfarin in control rats. Warfarin inhibition increased to greater than 90% in cytosols from 3MCA treated rats. Microsomal azoreductase and DT diaphorase were increased 4 and 2 fold respectively by 3MCA. These results indicate that riboflavin deficiency does not inhibit the induction of azoreductase and diaphorase by 3MCA. (no Refs)

13. METABOLISM OF AFLATOXIN B1 (AFB1), AFLATOXIN G1 (AFG1) AND VITAMIN C INTAKE BY GUINEA PIG LIVER PREPARATION IN VITRO.

Domngang F, Emerole G
Service de Biochimie, Universite de Yaounde, B.P. 812, Cameroun, West Africa
Biochem Pharmacol; 31(14):2327-2330 1982

Male and female guinea pigs weighing 150-200 g were divided into three groups, with equal number of males and females in each group. They were fed an experimental diet which varied as follows: Group I, 0 mg vitamin C/g of diet; Group II, 1.08 mg/g and Group III, 5.4 mg/g, for 28 days. Twenty-four hr after the last feeding, liver slices and 9,000 g supernatant were prepared from each group, according to sex, and used for enzyme assays. For the demethylation assay, enzyme activity expressed as amount of formaldehyde produced from AFB1 or AFG1/hr/g fresh liver was seen to increase with the two levels of ascorbic acid intake in females. Males showed an enhancement of activity only in Group II and remained with the same production of formaldehyde as above in Group III. Although in each dietary group, the activity was higher in males than in females the variation in the amount of formaldehyde produced from one group to another was higher with females than with male guinea pigs. However with both sexes, the production of formaldehyde from AFG1 was greater than from AFB1. For the hydroxylation assay, enzyme activity was expressed as amount of metabolites (a) and (b) produced. Compared to Group II, which offered a control level of ascorbic acid, Group I fed without vitamin C showed a decreased production of metabolite (a) with males and females. Moreover, high intake of ascorbic acid in Group III decreased the production of metabolite (a) and (b) in males, while in female guinea pigs the reduction was observed only with (b). (Author abstract) (25 Refs)

14. VITAMINS A, C AND E IN RELATION TO LUNG CANCER INCIDENCE (MEETING ABSTRACT).

Lopez-S A, LeGardeur BY
Department of Medicine, Louisiana State University School of Medicine, New Orleans, LA
Am J Clin Nutr; 35(4):851 1982

In previous reports, we have shown an inverse relationship between lung cancer incidence and vitamin A (VITA) and B-carotene (BCAR). Currently, we are expanding our study to evaluate if vitamin C (VITC) and vitamin E (VITE) are also associated with lung cancer incidence. Nutrient levels were studied in 29 persons newly diagnosed, by positive tissue or cytology report, with primary lung carcinoma (LC) and 29 hospitalized persons (H) serving as controls. The controls were matched by age, race, sex, and place of residence (parish). The data suggest that a possible interrelationship exists between vitamins A, C, E and incidence of lung cancer. The nature of this relationship needs to be established. (2 Refs)

GENERAL NUTRITIONAL ASPECTS OF CARCINOGENESIS

15. BREAST CANCER AND SERUM CHOLESTEROL.

Hiatt RA, Friedman GD, Bawol RD, Ury HK
Dept. Medical Methods Res., Kaiser-Permanente Medical Care Program, 3451 Piedmont Ave., Oakland, CA, 94611
JNCI; 68(6):885-889 1982

Recent studies have suggested a role for dietary fat in the etiology of breast cancer. The relation of serum cholesterol and other serum lipid measures to breast cancer incidence was investigated in a cohort of 95,179 women who had multiphasic health checkups from 1964 through 1972. Through 1977, 1,035 new breast cancer cases occurred in over 752,000 person-yr of follow-up. Age-adjusted incidence rates were 1.45, 1.37, 1.31, and 1.40/1,000 person-yr from the lowest to the highest quartile of serum cholesterol level, respectively. Similarly, no statistically significant relation was detected between beta-lipoprotein of total lipids and breast cancer. The sample size was sufficiently large to have detected a relative risk of 1.4 or larger with a probability of 99.9% at the 0.05 level of significance. The expected relation of breast cancer to established risk factors was confirmed by univariate analysis, and serum cholesterol and breast cancer were not associated after simultaneous consideration of nine other risk factors by multivariate analysis. These data suggest that the postulated causal relation between dietary fat and breast cancer does not act via an effect on circulating lipid levels. (Author abstract) (37 Refs)

16. ENHANCEMENT OF BOWEL CARCINOGENESIS BY DIETARY CHOLESTEROL INDEPENDENT OF BILE ACIDS AND DURING TUMOR INITIATION IN THE RAT (MEETING ABSTRACT).

Broitman SA, Gottlieb LS, Vitale JJ
Boston University School of Medicine, Boston, MA
Am J Clin Nutr; 35(4):842 1982

Demographically, cholesterol intake is directly related to the incidence of colon cancer. Experimentally, dietary cholesterol augments bowel carcinogenesis; lowering serum cholesterol levels by diet increases the yield of bowel tumors compared to rats with elevated serum cholesterol levels. The following was undertaken to ascertain if the effects of dietary cholesterol on bowel carcinogenesis is 1) related to the fecal excretion of bile acids, and 2) manifest during the initiation or promotional stage. Sprague-Dawley rats were fed a basal semi-synthetic diet containing 20% coconut oil. Supplementing this basal diet with 1% cholesterol increased the yield of 1,2 dimethylhydrazine induced bowel tumors from 1.83 to 3.17 tumors/rat. Cholic acid, 0.3%, added to the basal diet or the cholesterol diet did not increase tumor yields over those not fed cholic acid. In part two, a direct acting carcinogen, N-methyl nitrosourea (NMU), was given intrarectally. When cholesterol was added to the basal diet and fed throughout 40 weeks, large bowel tumor yield was increased from 1.87 (basal diet) to 3.69 (cholesterol diet) large bowel tumors/rat. Feeding rats the basal diet for 10 weeks (during which time NMU was administered) followed by feeding the cholesterol diet for 30 weeks did not increase tumor yield over those fed the basal diet alone. (no Refs)

17. EFFECT OF DIET ON PLASMA AND URINARY HORMONES IN SOUTH AFRICAN BLACK MEN WITH PROSTATIC CANCER.

Hill P, Wynder EL, Garbaczewski L, Walker AR
Dept. Surgery, Medical Sch., Univ. Cape Town, Observatory 7925, Cape Town, South Africa
Cancer Res; 42(9):3864-3869 1982

Epidemiological evidence suggests that the incidence and death rate from prostatic cancer, an endocrine-associated disease, are related to environmental factors including diet. In this study, a comparison of serum and urinary levels of steroid hormones was carried out in healthy, elderly, rural South African black men on a vegetarian diet (a low-risk population), and a comparable group of men with prostatic cancer. In these prostatic cancer patients, plasma androgen levels decreased, while estrogen levels increased. Concomitantly, the androsterone:etiocholanolone ratio increased, and a greater proportion of estrogens was excreted as estriol. When transferred to a Western diet, plasma androgens showed a further decrease and a greater increase in estrone in prostatic cancer patients. In prostatic cancer patients, the total urinary androgen and estrogen levels were unaltered. However, in elderly healthy men, the Western diet decreased the excretion of estrogens and androgens. Thus, a Western diet supplemented the decrease in plasma androgens initially present in these patients. Evidence suggests that the decrease in plasma androgens increases the estrogen:androgen ratio, which may lead to hyperplasia of the prostatic ductal epithelia, a change enhanced by a Western diet. Changes in urinary steroid hormone levels in South African black patients comparable to those reported in white prostatic cancer patients indicate that hormonal changes must be related to several environmental factors, apart from diet. A simultaneous study of the steroid hormone composition of blood and prostatic fluid in this low-risk population is suggested. (Author abstract) (54 Refs)

18. RESEARCH TECHNIQUE FOR EPIDEMIOLOGIC INVESTIGATION OF NUTRITION AND CANCER.

Moore JV, Prestridge LL, Newell GR
Dept. Cancer Prevention, Univ. Texas M.D. Anderson Hosp. and Tumor Inst. at Houston, 6723 Bertner Ave., Houston, TX, 77030
Nutr Cancer; 3(4):249-256 1982

When investigating cancer etiology, epidemiologists often correlate estimates of current dietary practices to cancer incidence. There is evidence that diets may be basically stable over time due to deep-rooted habits; however, changes in eating behavior are often associated with life-style or health-status changes. By using a diet-related life-event schedule, dietary changes resulting from life events may be more easily recalled and a more accurate characterization of diet patterns during neoplasm initiation may be made. (Author abstract) (66 Refs)

19. THE EFFECTS OF DIET AND CARCINOGEN ON THE NATURAL KILLER CELL ACTIVITY OF SPRAGUE DAWLEY RATS.

Longley RE
The University of Oklahoma, Norman, OK
Diss Abstr Int [B]; 42(11):4355-B 1982

This study was conducted to determine if dietary fat and/or administration of 7,12 dimethylbenz(a)anthracene (DMBA) to Sprague Dawley rats affected the natural killer (NK) cell activity of these animals. The inhibitory activity of NK cells was dependent on the quantity of fat in the diet of the rats from

which these cells were obtained. Rats which were fed a 20% corn oil diet (high fat) generally had lower levels of inhibitory activity against YAC-1 tumor cells compared to rats fed a diet containing 2% linoleic acid (low fat). While choline deficiency or treatment with DMBA did not alter NK cell activity in either dietary group, the combination of both factors caused a significant reduction in NK cell activity when rats were maintained on a high fat diet. The proliferative activity of NK cells were generally higher in rats on the high fat diet compared to rats on the low fat diet. NK-induced inhibition of YAC-1 cell proliferation correlated inversely with the proliferative activity of the NK cells. Some of the rats fed high fat diets had NK cells whose proliferative activity was approximately twice that of rats fed low fat diets, and whose corresponding inhibitory activity against YAC-1 tumor cells was one-half that of rats on low fat diets. NK cell activity was not constant during the period of study, but tended to display cyclic responses, particularly in rats in the high fat diet. There was no difference in the level of inhibitory activity of NK cells from rats on a low fat diet compared to rats on the high fat diet, when the R2T2 tumor cell line was used as the target cell. While the ability of NK cells to inhibit proliferation of YAC-1 tumor cells in vitro was diet dependent, the inability to show diet dependency on the ability of NK cells to inhibit the proliferation of R2T2 tumor cells in vitro did not necessarily rule out an in vivo role for NK cells in the resistance of rats to DMBA-induced tumors. (no Refs)

20. IMMUNE REACTIONS IN INADEQUATE NUTRITION AND IN AFLATOXIN CARCINOGENESIS.

Popov AA, Krystev LP, Kamenova BB
Inst. Gastroenterology and Nutrition, Sofia, Bulgaria
Vopr Pitan; (3):58-60 1982

Relationships between inadequate nutrition, humoral immunity, and incidence of aflatoxin-induced tumors were studied in male Wistar rats. The aflatoxin was administered ip, 10 ug/rat, 5x/wk. Rats in Group 1 received standard nutrition, while rats in Group 2 received a reduced ration. Colonic leiomyosarcomas, but no hepatomas, were observed to develop. The incidence of the sarcomas in the Group 2 rats was significantly lower than that in the Group 1 rats. The reduced incidence of aflatoxin-induced tumors in the Group 2 rats was shown to be associated with immunological deficiency. It was assumed that the immune deficiency was accompanied by inhibition of enhancement in the course of aflatoxin carcinogenesis. (14 Refs) (In RUS)

21. EFFECT OF N-(4-HYDROXYPHENYL)RETINAMIDE ON FOOD INTAKE AND GROWTH IN RATS (MEETING ABSTRACT).

Radcliffe JD, Moon RC
Illinois Institute of Technology Research Institute, Chicago, IL, 60616
Fed Proc; 41(3):357 1982

N-(4-hydroxyphenyl)retinamide (HPR) has an anti-proliferative effect on rat mammary epithelium that may be involved in HPR inhibition of mammary carcinogenesis. Body weight gain is reduced during the first week of HPR feeding. An experiment was initiated to determine whether this is due to reduced food intake and if so, how this might modify the effect of HPR on the mammary gland. Retinoid levels in mammary gland and body fat were also determined. Female SD rats (mean wt 176g) were used. Group A was fed, ab lib, a diet containing HPR (782 mg/kg diet). Groups B and C were fed placebo diet: B was fed ab lib and C was pair-fed to A. From age 58-65 days, food intake was lower for A (12.6g/rat/day) than for B (17.3g/rat/day). Group A failed to gain weight, but B gained 19g. After 66 days, food intake was similar in both groups. Final mean body weight (215 days of age) was similar for all groups (283-288g). Mammary glands of groups B and C were similar, but glands of group A showed decreased ductal branching and end bud proliferation. HPR levels in mammary gland and perirenal fat were 10.5 and 21.0 mg/g tissue, respectively. Initial reduction in body weight gain in HPR fed animals is due to reduced food intake which does not affect the anti-proliferative effect of HPR on mammary epithelium. (no Refs)

22. DRUG-NITRITE INTERACTIONS: EFFECTS OF FLAVONOID FOOD CONSTITUENTS ON NITROSAMINE FORMATION (MEETING ABSTRACT).

Rao GS, Osborn JC, Adatia MR
Research Institute, American Dental Association Health Foundation, Chicago, IL, 60611
16th Annual Meeting of the Great Lakes ACS Region, June 7-9, 1982, Normal, Illinois. American Chemical Society 101 pp., 1982.

The formation of carcinogenic N-nitrosamines from the interaction of certain orally administered amino drugs with dietary and endogeneous nitrite in humans is currently under active investigation. Such nitrite-mediated toxification of drugs may occur in the stomach, mouth, intestine, bladder, and urogenital tract at a rate that is dependent on their structure (pka), reaction conditions (pH and presence of other ions), and nitrite concentration. Recent studies indicate that certain common food constituents act as inhibitors or catalysts of nitrosation reaction (eg, ascorbic acid is an inhibitor, while chlorogenic acid is a catalyst). In the present paper, we have evaluated the effects of the following flavonoids on drug nitrosation: rutin (1) and quercetin (2) (found in buck-wheat), and naringin (3) and naringenin (4) (found in grapefruit). All compounds were tested (0.5-10 mM) by incubating with drugs (aminopyrine, oxytetracycline or piperazine, 10 mg/ml) and sodium nitrite (20 mg/ml) (pH 1-5) at 37 C for 1 hour. Nitrosation of drugs was monitored by HPLC (high performance liquid chromatography). Flavonoids I and II, which contain catechol group, were found to inhibit drug nitrosation (up to 55%, depending on pH and concentration), while those without catechol group (III and IV) had no effect on nitrosamine formation. (no Refs)

23. EFFECT OF DIET ON THE METABOLISM OF 1,2-DIMETHYLHYDRAZINE (DMH) IN THE RAT (MEETING ABSTRACT).

Glauert HP, Bennink MR
Michigan State University, East Lansing, MI, 48824
Fed Proc; 41(3):356 1982

Dietary fat and liver have been shown to influence DMH-induced colon carcinogenesis in experimental animals. One possible mechanism is that diet alters DMH metabolism. In this experiment, we examined the effect of dietary fat, wheat bran, and agar on 1) the metabolism of DMH to products which bind to DNA in rat colon epithelial cells (CEC), and 2) the clearance of DMH and its metabolites from blood. Diets were fed for four weeks; labeled DMH was then injected ip and rats were killed at various time intervals (12-96 hours). CEC were isolated from the colon and DNA was purified from CEC. Maximum binding DMH metabolites to DNA was seen 36 hours after the injection of DMH. The amount of 14C binding to DNA was not altered by diet but a diurnal variation was evident. Maximum 14C binding at 9 pm was 32-109% greater than 14C binding at 9 am. Serum radioactivity was greater in

rats fed the high-fat diet and lower in rats fed the wheat bran compared to the control group. 14C disappearance from serum fit a 'two-pool model' with half-life for the first pool ranging from 27 to 33 hr and half-life for the first pool ranging from 62 to 71 hr; diet did not alter half-life for either pool. This experiment shows that diet does not alter the amount of DMH metabolites bound to DNA in CEC even though diet does change the level of DMH plus its metabolites in serum. It appears that there is a marked diurnal variation in DMH metabolism. (no Refs)

24. FAECAL MUTAGENS FROM POPULATION GROUPS WITHIN NEW ZEALAND AT DIFFERENT RISK OF BOWEL CANCER (MEETING ABSTRACT).

Ferguson LR, Alley PG
Medical School, University of Auckland, Auckland, New Zealand
Twelfth Annual Meeting of the European Environmental Mutagen Society, June 20-24, 1982, Dipoli, Espoo, Finland. European Environmental Mutagen Society 250 pp., 1982.

New Zealand Europeans have one of the world's highest incidences and age-adjusted death rates of bowel cancer. The rates for Polynesians (and Maoris), who live in a similar environment to the Europeans, are considerably lower. The two population groups consume different types and quantities of food, suggesting that dietary factors may account for the observed differences. We have sought feces from bowel cancer patients before therapy, and assayed either soluble mutagens from these. The data have been compared with similar samples from age and sex matched groups of non-symptomatic Europeans, Seventh Day Adventists (vegetarians), Maoris and Polynesians. There is no suggestion of a mutagen in Maori or Polynesian groups, whereas each European group shows significant mutagen levels. The highest values are reached in the bowel cancer patients, lowest in the Seventh Day Adventists. We suggest that there is a genetic component preventing formation of this mutagen in Maoris and Polynesians and that the lack of this mutagen might explain the very low bowel cancer incidence in this group. Diet apparently regulates the mutagen level in Europeans, but these data do not explain the exceptionally high incidence of bowel cancer in this group. (1 Ref)

25. PLASMA PROLACTIN (PRL) AND ITS METABOLIC CLEARANCE RATE (MCR) IN MALE AND FEMALE RATS FED 5 AND 20% DIETARY FATS (MEETING ABSTRACT).

Clinton SK, Mulloy AL, Visek WJ
University of Illinois, Urbana, IL, 61801
Fed Proc; 41(3):357 1982

It has been proposed that prolactin mediates the enhancement of mammary carcinogenesis by dietary fat. We have studied the effect of dietary fat level (10 and 40% of calories as corn oil) on plasma immunoreactive PRL and its MCR in male and female Sprague-Dawley rats. Rats were assigned to modified AIN diets at weaning. After 10 weeks, 15 rats per treatment were decapitated and trunk blood was assayed for PRL by radioimmunoassay. For the MCR studies 10 male and female rats per diet were anesthetized with pentobarbital, fitted with a jugular cannula and treated with 2-bromo-alpha-9 ergocryptine prior to receiving an exogenous bolus of PRL and collection of sequential blood samples to determine PRL clearance. There was no significant effect of fat on plasma PRL (ng/ml) in females at metestrus-diestrus [23 on low fat (LF) vs 17 on high fat (HF)] or proestrus-estrus [339 (LF) vs 329 (HF)]. There was also no significant difference in plasma PRL in male rats fed LF (39) vs those fed HF (55). Although male rats show a higher MCR than females there was no significant effect of fat. The MCR for males was .80 ml/min on LF and .76 mg/min on HF. The MCR for female rats was .36 and .42 respectively. The PRL and MCR data fail to show that dietary fat significantly changes the plasma PRL levels or its MCR. (no Refs)

26. NUTRITION AND DIET (MEETING ABSTRACT).

Newell GR
University of Texas System Cancer Center, M. D. Anderson Hospital and, Tumor Institute Houston, TX, 77030
American Cancer Society National Conference: The Primary Care Physician and Cancer, June 24-26, 1982, Washington, DC American Cancer Society; :A5 1982

During recent years there has been a growing awareness among both the oncologic and nutrition communities about an association between nutrition and cancer causation with the practical implication of manipulating nutrition as a means of preventing cancer. Epidemiologic correlations, prospective studies of selected populations, and dietary histories of cancer cases compared to suitable comparisons all point toward such associations. For some of the more common cancers in this country it is thought that individuals can exert a certain amount of control over his or her own cancer risk through diet and life-style. As more is learned about mechanisms of mutagens and carcinogens, it may be routinely possible to inhibit, block, or reverse the cellular processes involved in carcinogenesis. It has been estimated that up to 35% of cancers may eventually be prevented by dietary modification. Until the final answers are in, it seems prudent to reduce total dietary fat intake and increase total fiber intake. (no Refs)

27. DIETARY EFFECT ON GROWTH OF WALKER-256 TUMOR IN RATS (MEETING ABSTRACT).

Kwong E, Covey TR, Nesheim MC, Dills WL
Division of Nutritional Sciences, Cornell University, Ithaca, NY, 14853
Fed Proc; 41(3):356 1982

Free fatty acid diets with 20% glucose (FFA) and Commercial Laboratory Chow diet (CLC) were used to examine the effect of diet on growth of Walker-256 tumor in rats. Each diet was fed to control rats and rats inoculated with Walker-256 tumor cells. There were 6 rats in each experimental group and experiments lasted for 14 days. The weight gains and food intakes similar for control rats and tumor-bearing rats on the same diets. The rats fed CLC diet gained slightly more weight than the rats fed FFA diet but the difference was not statistically significant for either control or tumor bearing rats. The food intakes of rats fed CLC diet were significantly higher than the food intake of rats fed FFA diet for both control and tumor bearing rats. The rate of tumor growth in both dietary groups were very similar for 9 days post-transplant. The tumors of the rats fed FFA diet, continued to grow after this time, while the tumors of the rats fed CLC diet began to decrease in size after the 9th day-transplant. At the end of the experiment, tumors of the rats fed FFA were 13 to 17 grams and the tumors of the rats fed CLC diet were 1 to 5 grams. The result indicates that inhibitory effect of CLC diet on the growth of Walker-256 tumor carcinogenesis was exerted during the post-initiation phase. (no Refs)

28. ADAPTIVE EFFECTS OF DIETARY ETHANOL IN THE PIG: CHANGES IN PLASMA HIGH-DENSITY LIPOPROTEINS AND FECAL STEROID EXCRETION AND MUTAGENICITY.

Topping DL, Weller RA, Nader CJ, Calvert GD, Illman RJ
Div. Human Nutrition, CSIRO, Glenthorne Lab., O'Halloran Hill 5158, South Australia
Am J Clin Nutr; 36(2):245-250 1982

Six young mature male pigs were maintained on a high fat, low fiber 'Western' type diet. Substitution of ethanol for sucrose raised plasma total cholesterol, an increase that was solely due to a rise in high-density lipoproteins. Plasma triacylglycerols and apo-B concentrations were unchanged and although apo-A1 rose with ethanol, this was not statistically significant. Ethanol did not alter total fecal steroids but both bile acids and the ratio of bile acids/neutral sterols were increased. In fecal extracts from these animals, mutagenic activity in the Ames bacterial test was also raised. The data are discussed in relation to the relationships between dietary ethanol and coronary heart disease and colorectal cancer. (Author abstract) (30 Refs)

GENERAL REVIEWS AND OTHER STUDIES

29. ENVIRONMENTAL CARCINOGENS. JANUARY, 1979-FEBRUARY, 1981 (CITATIONS FROM THE NTIS DATA BASE).

National Technical Information Service
Springfield, VA
Environmental Carcinogens. January, 1979-February, 1981 (Citations from the NTIS Data Base). Available from National Technical Information Service, Springfield, VA, as PB82-807389:, 291 pp., 1982.

The bibliography cites research reports on the identification, sources, and toxicity of environmental carcinogens. Included are studies on pesticides, fire extinguishing agents, environmental pollutants, food additives, radiation, and other known or suspected carcinogenic substances. (This updated bibliography contains 285 citations, none of which are new entries to the previous edition). (Author abstract)

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NOTICE OF CURRENT REVIEWS: CARCINOGENESIS

As an additional service to readers of carcinogenesis (CK series) CANCERGRAMS, each issue now provides a bibliography of review articles of interest to carcinogenesis researchers, selected from journals screened for that month's CANCERGRAMS.

CHEMICAL CARCINOGENESIS

1. CURRENT APPROACHES TO THE EXPERIMENTAL INVESTIGATION OF CHEMICALS IN RELATION TO CANCER OF THE BRAIN.
Swenberg JA, Zimmerman HM, Maltoni C, Kurland L, Laerum OD, Kleihues P, Selikoff I, Heidelberger C
Chemical Industry Inst. Toxicology, Research Triangle Park. NC, 27709
Ann NY Acad Sci; 381:43-53 1982

2. PRODUCTION OF BRAIN TUMORS WITH AROMATIC HYDROCARBONS.
Zimmerman HM
Montefiore Hosp. and Medical Center, 111 East 210th St., Bronx, NY, 10467
Ann NY Acad Sci; 381:320-324 1982

3. ORAL CONTRACEPTIVE STEROIDS AND MALIGNANCY.
Grant E
No affiliation given
Clin Oncol; 8(2):97-102 1982

4. METAL CARCINOGENESIS - WHAT IS REAL (MEETING ABSTRACT).
Furst A
Institute of Chemical Biology, University of San Francisco, San Francisco, CA, 94117
Environmental Analytical Chemistry, Airborne Mutagens and Carcinogens, June 16-18, 1982, Provo, Utah. Electric Power Research Institute 44 pp., 1982.

5. GENETIC EFFECTS OF N-METHYL-N'-NITRO-N-NITROSOGUANIDINE AND ITS HOMOLOGS.
Gichner T, Veleminsky J
Inst. Experimental Botany, Czechoslovak Acad. Sciences, Flemingovo 2, 160 00 Praha 6, Czechoslovakia
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RADIATION CARCINOGENESIS

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